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Treatment of Congestive Heart failure

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Heart failure is defined as the inability of the heart to supply sufficient blood flow to meet the needs of the body. The term "congestive heart failure" implies that the impaired blood flow is causing fluid retention in the lungs, legs, ankles or feet. Other common symptoms include shortness of breath when lying down or during exercise, fatigue and weakness, reduced exercise capacity, and rapid or irregular heartbeat.

Coronary artery disease (atherosclerosis) and heart attack are the most common causes of heart failure along with high blood pressure, faulty heart valves, damage to the heart muscle, inflammation (myocarditis), and congenital heart defects. Untreated chronic heart arrhythmias, especially atrial fibrillation, may also lead to heart failure as may the presence of diabetes, severe anemia and thyroid problems. Finally, there is evidence that heart failure is associated with a deficiency of thiamine (vitamin B1) which is exacerbated with the use of thiazide diuretics.

The primary diagnostic markers of heart failure are left ventricular ejection fraction of less than 40% and a blood (plasma) level of brain natriuretic peptide (BNP) in excess of 100 pg/mL. An elevated blood (serum) level of C-reactive protein is also associated with heart failure.

Conventional Treatment

Regardless of the cause and manifestation of the disease (left-sided heart failure, right-sided heart failure, systolic heart failure or diastolic heart failure) the medications commonly prescribed for heart failure are as follows:

- ACE inhibitors such as enalapril, lisinopril and captopril which dilate blood vessels to lower blood pressure, improve blood flow, and decrease the workload on the heart.

- Angiotensin II receptor blockers such as losartan (Cozaar) and valsartan (Diovan) which have effects similar to those of ACE inhibitors.

- Beta-blockers such as bisoprolol and carvedilol which slow heart rate and reduce blood pressure.

- Digoxin (Lanoxin) which slows the heart beat and increases the strength of heart muscle contractions. Unfortunately, it has many serious adverse effects and is probably not that effective. See www.afibbers.org/resources/digoxin.pdf for further reading. It is now considered 5th line treatment for heart failure and recent research shows that best results

are obtained if the daily dose is reduced to 0.125 mg/day, or whatever dosage is required to produce a serum level of 0.5 – 0.9 ng/mL.[58] Digoxin toxicity is a major cause of hospital admissions and the risk of a toxic reaction is increased 3-fold with the commensurate use of diuretics.[59] Low potassium and treatment with statin drugs are also risk factors for digoxin toxicity.

- Diuretics such as butmetanide and furosemide which help prevent and eliminate fluid build-up.

- Aldosterone antagonists such as aldosterone and eplerenone. These are potassium-sparing and may help reverse scarring of the heart and help patients with severe heart failure live longer.

The most recent *AHA/ACCF Guidelines for the Management of Heart Failure*[1] recommend the following treatment protocol for patients with structural heart disease (valve problems) and symptoms of heart failure[2]:

- Treatment of hypertension if present
- Treatment of high cholesterol if needed
- Regular exercise
- No smoking and limited alcohol intake
- Restricted salt intake
- Routine drug therapy with diuretics, ACE-inhibitors, beta-blockers
- Selected drug therapy with aldosterone antagonists, angiotensin II receptor blockers, digoxin.

The following comments in the guidelines regarding potassium are of particular interest[3]:

> *"Patients with HF (heart failure) should be monitored carefully for changes in serum potassium, and every effort should be made to prevent the occurrence of either hypokalemia or hyperkalemia, both of which may adversely affect cardiac excitability and conduction and may lead to sudden death. Activation of both the sympathetic nervous system and renin-angiotensin system can lead to hypokalemia and most drugs used for the treatment of HF can alter serum potassium. Even modest decreases in serum potassium can increase the risks of using digitalis and antiarrhythmic drugs, and even modest increases in serum potassium may prevent the use of treatments known to prolong life. Hence, many experts believe that serum potassium concentrations should be targeted in the 4.0 to 5.0 mmol per liter range."*

Alternative Treatment

The goal of alternative and complementary therapies is to increase the pumping efficiency of the heart and to alleviate the adverse effects of conventional treatment. Several natural substances have been found effective in the treatment of heart failure. Substantial evidence of efficacy is available for the following:

- Coenzyme Q10
- Pycnogenol
- L-carnitine
- Thiamine
- Magnesium
- Potassium
- D-ribose
- Fish oil
- Hawthorn
- Vitamin D
- Arginine
- Taurine

Coenzyme Q10

Coenzyme Q10 (ubiquinone, ubiquinol) is an essential component of the mitochondria, the energy-producing unit of every cell of our body. Heart failure is associated with a pronounced coenzyme Q10 deficiency, and low coenzyme Q10 levels are associated with increased mortality in heart failure patients.[4,5] There are several clinical trials which clearly show that supplementation with coenzyme Q10 (150 – 650 mg/day) markedly improves heart function in heart failure patients.[6-9] More recent research has shown that ubiquinol, the reduced form of coenzyme Q10 is even more effective in the treatment of heart failure.[10]

Coenzyme Q10 supplementation is of extreme importance in heart failure patients on statin drugs. Research has shown that these drugs seriously impede the synthesis of coenzyme Q10 leading to such adverse effects as myalgia (muscle pain), fatigue, breathing difficulties, memory loss, and peripheral neuropathy. There is also growing evidence and concern that the indiscriminant use of statin drugs over the past decades is, to a large extent, responsible for the current epidemic of heart failure, which now claims more than 500,000 new victims a year in the United States alone.

Fortunately, supplementation with coenzyme Q10, preferably in conjunction with discontinuation of statin drugs, can completely reverse these effects.[11-14]

Well-functioning heart cell mitochondria are essential to heart health. Coenzyme Q10 is the "spark plug" that powers the mitochondria. Recently, a new supplement, pyrroloquinoline quinone (PQQ) has been developed which markedly increase the formation of new mitochondria.[15-17] Thus, it would seem that a protocol which combines ubiquinol (3 x 100 or 3 x 200 mg/day) with PQQ (20 mg/day) would be greatly beneficial.

Pycnogenol

Pycnogenol is a powerful antioxidant and anti-inflammatory extracted from the bark of the French Maritime pine tree. It has an amazing range of beneficial effects including reduction of glucose levels, management of chronic asthma, reduction of platelet aggregation (as effective as aspirin, but without the negative side effects), and regeneration of vitamins C and E. Of more immediate interest is a recent finding that pycnogenol, in combination with coenzyme Q10, materially improves the health of heart failure patients. An Italian clinical trial recently concluded that the combination of pycnogenol and coenzyme Q10 (50 mg/day Q10 and 15 mg/day pycnogenol) increased left ventricular ejection fraction and walking distance in a group of heart failure patients.[18]

Carnitine

Carnitine is a vitamin-like compound responsible for the transport of long-chain fatty acids into the mitochondria. Thus it, along with coenzyme Q10, is essential for cellular energy production. There is evidence that l-carnitine itself reduces symptoms of chronic heart failure[19], but research into the benefits of carnitine supplementation has largely focused on propionyl-l-carnitine, a naturally occurring derivative of l-carnitine. Several clinical trials have concluded that treatment with orally administered propionyl-l-carnitine (3 x 500 mg/day) is effective in increasing exercise capacity and left ventricular ejection fraction in heart failure patients.[20-22] Not surprisingly, a combination of l-carnitine and ubiquinol has also been found effective in reducing breathlessness, fatigue and palpitations, and improving walking distance in heart failure patients.[23]

Thiamine

Thiamine, also known as vitamin B1, is a prominent member of the water-soluble B-complex. It is required for the proper metabolism of proteins, carbohydrates and fats, and is intimately involved in ATP production (energy generation) in every cell. Clinical research has shown that about a third of hospitalized heart failure patients are deficient in thiamine and that from 55 to 98% of patients on the diuretic furosemide suffer from severe thiamine deficiency.[24,25] Fortunately, it

is possible to reverse the adverse effects of thiamine deficiency by supplementing with 300 mg/day of thiamine.[26]

Magnesium

Magnesium is of key importance to human health. It participates in over 300 enzymatic reactions in the body. A deficiency has been linked to conditions such as irregular heartbeat, asthma, emphysema, cardiovascular disease, high blood pressure, mitral valve prolapse, stroke and heart attack, diabetes, fibromyalgia, glaucoma, migraine, kidney stones, osteoporosis, and probably many more. About 99% of the body's magnesium stores are found in the bones and tissues and heart tissue is particularly rich in this important mineral. Only 1% of the body's magnesium is actually present in the blood so a standard blood analysis is a very poor way of determining overall magnesium status.

Magnesium deficiency is widespread in the general population and especially pronounced in atrial fibrillation and heart failure patients, especially if treated with loop diuretics (thiazides), digoxin and ACE inhibitors.[27,28] There is evidence that magnesium deficiency is associated with a much lower survival rate in heart failure patients.[29] Fortunately, there is also evidence that replenishment of magnesium with oral supplementation, specially magnesium orotate, can markedly improve both clinical symptoms, survival and quality of life.[30]

A growing body of evidence points to a close connection between magnesium deficiency and mitral valve prolapse and, perhaps even more importantly, clinical trials have shown that supplementation with magnesium can partially or fully eliminate the symptoms of mitral valve prolapse.[31,32]

Intramuscular injections of magnesium sulfate and oral supplementation with chelated magnesium (magnesium glycinate) are effective means of increasing magnesium level in heart cells.

Potassium

Potassium is a very important electrolyte and an adequate level is essential to ensure proper heart function. As in the case of magnesium, potassium deficiency (hypokalemia) is widespread among heart failure patients and is further exacerbated if the patient is on loop diuretics (thiazides), digoxin and ACE inhibitors.[28] Scottish researchers have found that the optimum potassium level for heart failure patients is between 4.5 and 5.5 mmol/L (mEq/L). Levels lower than this increase the risk of ventricular arrhythmias and death. For those with low potassium levels, the researchers recommend supplementation with potassium and magnesium combined with aldosterone blockade to prevent increased potassium excretion.[33] Aldosterone blockade can be achieved through the use of ACE inhibitors, angiotensin II type 1 receptor blockers, or aldosterone receptor blockers (spironolactone and eplerenone). Excessive potassium excretion can also be prevented through the use of potassium-sparing diuretics such as triamterene (Dyrenium) and amiloride (Midamor).[34]

Whichever protocol is used to achieve a potassium level between 4.5 and 5.5 mmol/L, it should be kept in mind that a low magnesium level (hypomagnesemia) increases potassium excretion, and it is very difficult to remedy hypokalemia without first attaining normal magnesium levels. One study found that 42% of people with low magnesium levels also had low potassium levels.[35,36]

D-ribose

D-ribose is a simple, five-carbon sugar which acts as fuel in the production of ATP, the body’s source of energy. Clinical studies have shown that d-ribose is highly effective in increasing ATP production in heart failure patients and thus ameliorating symptoms of fatigue, improving the heart’s pumping capacity, and generally resulting in a better quality of life.[37] Two clinical trials have found that supplementation with 5 grams of d-ribose 3 times daily is effective in improving heart failure symptoms.[38,39]

Fish oil

There is overwhelming evidence that consumption of fatty fish and supplementation with fish oil are highly beneficial in maintaining heart health. A fish oil intake of at least 1 gram/day reduces the risk of sudden cardiac death by as much as 80%, most likely through the ability of fish oil to increase heart rate variability, which is usually too low in heart failure patients.[40] The large GISSI-HF clinical trial found that supplementation with 1 gram/day of fish oil reduced hospital admissions and death in a group of 7000 heart failure patients.[41] A more recent trial involving 133 heart failure patients concluded that supplementation with fish oil increases left ventricular ejection fraction and exercise capacity, and reduces annual hospitalization rate from 30% to 6%.[42]

Hawthorn

Hawthorn (*Crataegus oxyacantha*) is a powerful heart tonic widely used in Germany in the treatment of heart failure, either on its own or in addition to standard medical treatment. Hawthorn increases the strength of the heart's contraction (inotropic effect similar to that exhibited by digoxin). It also increases blood flow in the heart, increases left ventricular ejection fraction and exercise tolerance, and relieves other symptoms of heart failure. The German Commission E has approved the use of hawthorn in stage II (NYHA classifi8cation) heart failure.

The product most widely used in Germany is WS1442 which is an extract of hawthorn leaf and flower standardized to contain 18.75% of oligomeric procyanidins. A recent Cochrane review of 10 clinical trials evaluating the effect of hawthorn in heart failure patients concluded that supplementation with hawthorn (most likely 450 mg of WS1442 twice daily) improved exercise tolerance and significantly reduced symptoms such as shortness of breath and fatigue. Most of the clinical trials used hawthorn as an adjunct to standard medical treatment. Adverse effects were infrequent, mild and transient. The Cochrane researchers conclude that "*there is a significant benefit in symptom control and physiologic outcomes from hawthorn extract as an adjunctive treatment for chronic heart failure*".[43]

Vitamin D

Vitamin D is not really a vitamin, but rather a hormone which the body can make using sunlight. The skin contains a cholesterol derivative, 7-dehydrocholesterol (provitamin D), which is converted to vitamin D when exposed to sunlight. Vitamin D is converted in the liver to 25-hydroxyvitamin D [25(OH)D] which in turn is converted, mostly in the kidneys, to the active hormone $1,25(OH)_2D$ or calcitriol. There are two forms of vitamin D supplements – **vitamin D3** or cholecalciferol and **vitamin D2** or ergocalciferol. Vitamin D2 is synthetic and has only about half the efficacy of vitamin D3 when it comes to raising blood levels of 25(OH)D, the commonly used measure of vitamin D concentration.

Vitamin D deficiency is widespread and has been implicated in cancer, osteoporosis, hypertension, diabetes, rheumatoid arthritis and multiple sclerosis. Most researchers now consider a 25(OH)D level below 50 nmol/L (20 ng/mL) to be deficient and an optimum level to be about 75 nmol/L (30 ng/mL). A low vitamin D [25(OH)D] level is common among heart failure patients and is an indicator of a poor prognosis. Dutch researchers have found that heart failure-related mortality increases by 10% for each 10 nmol/L decrease in 25(OH)D level.[44] Fortunately, it is relatively simple to correct a vitamin D deficiency. It can be achieved slowly through oral supplementation with 2000 to 4000 IU/day of cholecalciferol over a 6-month period, or quickly by using one-time doses as high as 500,000 IU.[45,46]

Arginine

L-arginine is a semi-essential amino acid that acts as a physiological precursor of nitric oxide. Nitric oxide, in turn, plays a crucial role in regulating blood circulation, dilates blood vessels, and helps prevent the formation of blood clots. The effect of supplementation with arginine has been studied extensively and it has been found useful in the prevention and treatment of cardiovascular disorders including mild and moderate heart failure.[47] Supplementation with l-

arginine has been found to increase exercise tolerance and improve right ventricular ejection fraction in heart failure patients.[48-50] Improvement may be seen in as little as 7 days using dosages of 2 to 3 grams three times daily.

Taurine

Taurine is an amino acid widely distributed in human tissue. It is essential for proper cardiovascular function, and the development and function of the central nervous system, retina and skeletal muscle. It is a powerful antioxidant and protects against toxicity of lead and cadmium. It has also been found effective in lowering cholesterol and by keeping potassium and magnesium inside of heart cells and excessive sodium out, it helps prevent arrhythmia (including atrial fibrillation), and acts as a diuretic.

Taurine deficiency is common among heart failure patients; thus, it is not surprising that Japanese researchers, 30 years ago, reported that taurine supplementation (2-3 grams/day) is effective and entirely safe in the treatment of congestive heart failure.[51-54] More recent research has shown that taurine supplementation (500 mg three times daily) for 2 weeks significantly increases exercise capacity in heart failure patients.[55] There is also evidence that taurine exerts an inotropic effect similar to that of digoxin (without the side effects), and that it has diuretic effects and counteracts the adverse effects of angiotensin II.[56,57] Thus taurine supplementation could potentially reduce the need for treatment with ACE inhibitors/angiotensin II receptor blockers and digoxin.

Summary

It is clear that heart failure patients are often deficient in nutrients crucial to proper heart function. In many cases, these deficiencies are exacerbated by drugs (digoxin, diuretics, statins and ACE inhibitors) prescribed as part of the standard medical treatment for heart failure. It is thus of utmost importance that patients

- confirm that their medications are indeed needed and that dosages are optimum – minimizing, or even better, eliminating digoxin is particularly important.
- determine when possible if they are deficient in any of the critical nutrients discussed.
- rectify confirmed and likely deficiencies with appropriate supplementation.
- gradually wean off redundant medications as their condition improves as a result of the elimination of nutrient deficiencies.

The supplements mentioned in this report can all be ordered through the afibbers.org vitamin shop at http://www.afibbers.org/vitamins/vitamin14.htm

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