

Understanding Medical Treatment of Heart Failure

Heart failure contributed to almost 300,000 deaths in 2006 and costs the American health care system almost 39.2 billion dollars annually. Approximately, 20% of people die within one year of diagnosis. It affects almost 5.8 million Americans and is newly diagnosed in almost 670,000 people each year (1).

Heart failure is most commonly caused by chronic hypertension, myocardial infarction, valvular heart disease or cardiomyopathy. Other factors related to heart failure include: ischemic heart disease, obesity, diabetes, physical inactivity, excessive alcohol intake, cigarette smoking, chemotherapeutic agents and certain toxins (see Table 1).

Heart failure is an inability of the heart to pump out enough blood to meet the demands of the body. The ineffective heart is unable to get adequate oxygen to the body resulting in the symptoms of heart failure. To compensate for the failing heart the body activates the sympathetic nervous and renin-angiotensin-aldosterone systems (RAAS). The sympathetic nervous system increases blood pressure, heart rate and cardiac afterload while the RAAS increases sodium retention – which helps the body compensate to meet metabolic demands during the acute event, but over the long-term contributes to the progressive nature of heart failure. Treatment options are partially focused on stopping the negative impact of the activated nervous and endocrine systems to modulate the long-term course of the disease.

Systolic versus Diastolic

Heart failure is classified as systolic or diastolic. By determining ejection fraction, an echocardiogram can determine which type of heart failure is present. Ejection fraction

is the measure of the amount of blood ejected from the heart with each beat. When the heart is unable to eject a normal percentage of blood the ejection fraction is less than 40-50% and heart failure is classified as systolic. When the heart does not fill properly with blood, but the percentage of blood ejected with each beat is normal, then diastolic heart failure is diagnosed. Diastolic heart failure is associated with lower death rates than systolic HF though its affect on quality of life is similarly dire. Diastolic failure has similar post-hospital mortality risk and re-hospitalization rates when compared to those with systolic heart failure (6).

Signs and symptoms

The signs and symptoms of heart failure (see Table 2) are related to increased amount of fluid in the body. The main symptoms of heart failure include: shortness of breath, edema, fatigue and orthopnea (difficulty breathing when lying on the back). In heart failure the blood backs up and overloads the lungs and venous system. The malfunctioning heart allows extra fluid to back up into the lungs contributing to shortness of breath and crackles in the lungs. As the fluid fills the venous system, fluid leaks out of the blood vessels commonly causing edema in the legs or abdomen. A combination of fluid in the lungs and the hearts inability to pump out enough oxygen to the working muscles results in fatigue. Gradual weight gain is another common consequence of heart failure. Other symptoms of heart failure include cough at night, intolerance to cold, wheeze and poor appetite.

Signs of heart failure include crackles in the lungs, an S3 gallop, edema, bulging neck veins, weight gain, elevated heart and respiratory rate, decreased oxygen levels, hepatomegaly, ascites and displaced point of maximal impulse.

Diagnosis

History and physical exam are the first steps in diagnosing heart failure. When the clinician is suspicious of heart failure diagnostic testing is initiated.

Blood tests including - complete blood counts to check for anemia or infection, kidney and liver function tests, electrolytes, thyroid test, B-type natriuretic peptide (BNP), and cardiac enzymes – are typically ordered. Blood tests can help determine if anything else is contributing to, causing or complicating heart failure.

The stressed left ventricle, like in decompensated heart failure, secretes BNP, a cardiac neurohormone. Levels less than 100 pg/ml make it unlikely that heart failure is present and levels over 400 pg/ml make the diagnosis likely.

Diagnostic testing can further define heart failure. Chest X-rays helps rule out pneumonia as well as determine if there is any fluid in the lungs or if the heart size is increased. Electrocardiograms can help diagnose any arrhythmias, old myocardial infarctions, left ventricle hypertrophy or ischemic changes which are causing or contributing to heart failure. Echocardiogram is an ultrasound of the heart, which evaluates how effective the heart is beating and if there are any problems with the heart valves. Stress tests or cardiac catheterizations are done if lack of blood flow to the heart is suspected, as this is a common cause or contributing factor to heart failure.

Treatment

Heart failure can be classified as acute and chronic. Acute heart failure is a sudden onset of signs and symptoms where the heart is not pumping out enough blood to supply the body with oxygen. Many of these patients are seen in the emergency room with the complaint of difficulty breathing and swollen legs.

The under functioning heart does not supply adequate blood flow to the vital organs of the body, which, overtime, damages the body. In response to the failing heart, the body tries to compensate by excreting catecholamines, hormones and activating the nervous system which causes the heart to change in a negative ways, in a process called remodeling. Treatment of chronic heart failure, which includes normalizing the catecholamines, hormones and nervous system, is essential to prevent damage being done to the body.

Acute Heart Failure

The first step in treating acute heart failure is to rid the body of extra fluid, which is causing the majority of distress. Diuretics clear the body of extra fluid by increasing urine output and are a common treatment of acute heart failure.

Depending on the severity of the heart failure, hospitalization may be required to provide oxygen, monitor blood chemistries and heart rhythm. Aggressive workup are typically undertaken to determinate the underlying cause of heart failure including a work up to rule out myocardial ischemia or infarction, hypertension, thyroid disease, kidney disease, arrhythmias, anemia, and infections.

Chronic Heart Failure

The main goals for treatment of chronic HF includes relief of symptoms, improving quality of life, maintaining independence, improving ability to function, preventing hospitalization, preventing exacerbations of acute heart failure, prolonging life and preventing the negative affects of an under performing heart.

Non-Drug Treatment

The main components of non-drug treatments include: exercising, salt restriction,

stopping smoking, weight loss, monitoring body weight and limiting alcohol.

Exercise training is a cornerstone in treating heart failure. It will not reverse the disease but increases physical function and improves quality of life. Exercise combats many of the effects of the disease, reduces deadly heart rhythms, slows down and may even reverse muscle wasting and improves blood flow.

Dietary changes can have a profound impact on heart failure. Ingestion of sodium increases fluid retention so it is important that those with heart failure limit the amount of sodium in the diet. Heart failure patients may notice that the day after ingestion of a large sodium meal, body weight increases. Eating high quantities of salt over consecutive days may result in an exacerbation of heart failure. Heart failure patients should not exceed 2-3 grams of sodium a day.

Toxic habits can have negative effects on the heart. Smoking cigarettes initiates a chemical cascade that leads to constriction of the blood vessels, elevated blood pressure and worsening of heart function. Not smoking is a key component to treating heart failure. Alcohol can have dire effects on the heart. Certain types of heart failure are directly related to the alcohol ingestion. Not all heart failure is caused by alcohol, but its intake should be limited, if not avoided, in anyone with heart failure.

Monitoring weight is a very subtle way to observe heart function. When the heart is not functioning optimally, fluid backs up in the body instead of being urinated out. Consequently, body weight increases. Therefore, checking weights daily helps to know if extra diuretics or an adjustment in treatment is needed.

Drug treatment

The mainstay of treatment in chronic, systolic HF is angiotensin converting enzyme

inhibitors (ACE-I) and beta-blockers (BB). Diuretics are primarily used to prevent excessive fluid build up and may not be needed by all patients. Digoxin, which was once the main treatment option for systolic heart failure is not used as universally. Aldosterone antagonists should also be considered in patients with severe symptoms.

Diuretics

Diuretics are a first-line treatment for acute heart failure and are used in some patients with chronic heart failure to prevent fluid buildup. Loop diuretics (e.g. Furosemide and Torsemide) are typically more effective than thiazides (hydrochlorothiazide). Diuretics are often only used during an acute attack and some patients do not require them for long-term treatment. It is best to avoid the use of diuretics in chronic heart failure as they have not been shown to improve mortality (2) and they negatively affect the production of chemicals that lead to progression of heart failure. Although, many patients with chronic heart failure need them to prevent fluid build up.

Side effects of diuretics include dehydration, dizziness, abnormal electrolytes and incontinence. Most troubling is that diuretics activate the RAAS that is associated with disease progression. If diuretics are being reduced or discontinued than, daily weights should be monitored to assure fluid is not accumulating. While on diuretics electrolytes, kidney function and glucose levels should be monitored.

Angiotensin Converting Enzyme Inhibitors/Angiotensin Receptor Blockers

Angiotensin II is a hormone that causes progression of the heart failure. Lowering the levels of angiotensin II slows the progression of heart failure and profoundly affects the natural course of the disease. Angiotensin II constricts blood vessels resulting in

elevated blood pressure. Constricted blood vessels make the heart work harder as it is working against more pressure. Therefore, high blood pressure leads to a faster progression of the disease.

Angiotensin Converting Enzyme inhibitors (ACE-I) and Angiotensin Receptor Blocker (ARB) are two medications that reduce death rates and improve cardiac function in heart failure. ACE-I prevent the formation of angiotensin II and therefore dilate the blood vessels and lower blood pressure. While ACE-I are considered the gold standard drug for heart failure ARB are a good alternative for those unable to tolerate ACE-I.

Patients with CHF have high levels of angiotensin circulating in their bodies. By using ACE-I the levels of angiotensin are reduced which decrease the negative effects angiotensin has on the progression of the disease. Angiotensin Receptor Blocker (ARB) are drugs that work very similar to ACE-I. They directly block the effect of angiotensin on the blood vessel walls and result in lower blood pressure.

Side effects of ACE-I include: low blood pressure, dizziness, high potassium, cough, stomach upset, headache and renal impairment. Reasons exist not to be on ACE-I including renal artery stenosis and propensity to side effects (e.g., worsening electrolyte imbalance, low blood pressure and cough). Monitoring electrolytes and kidney function is important as these medications can worsen renal function and potentially cause life-threatening hyperkalemia. Cough is a very common side effect of ACE-I, which at times makes the drug unbearable. ARB is commonly substituted for ACE-I when cough becomes bothersome.

Side effects of ARB are similar to ACE-I except they lack cough as a significant side

effect.

Beta Blockers (BB)

BB such as metoprolol, bisoprolol and carvedilol are another class of drugs used to treat both high blood pressure and heart failure. BB improve heart function, blood flow to the heart, symptoms and exercise tolerance. They also decrease death rates in those with congestive heart failure. One mechanism that they decrease death rates is by reducing life threatening abnormal heart rhythms that are common in heart failure.

These drugs do not show an immediate benefit. In fact, patients often feel worse when placed on these drugs. Improvement becomes apparent after a couple months on the drugs and those on the drug for a longer period of time see more improvements as the structure of the heart becomes positively remodeled.

These drugs should be started at a very low dose and the dose should be advanced slowly. This reduces the incidence of severe side effects. While BB have a profound impact on the disease close monitoring is essential. Blood pressure and weights should be monitored as some patients develop hypotension or worsening heart failure while on these drugs.

Common side effects of BB include: fatigue, dizziness, depression, stomach upset, slow heart rate and low blood pressure. Caution should be used with BB when certain diseases are present including diabetes, peripheral vascular disease, depression, asthma and chronic obstructive lung disease.

Aldosterone Antagonists

Spironolactone and eplerenone are the drugs in this class. Spironolactone reduces mortality, decreases hospitalization, and improves symptoms in those with stage IV

failure on standard therapy (3). The drug works by blocking the effects of a hormone called aldosterone. Aldosterone, when present in high quantities for a long period of time, changes the structure of the heart and accelerates the process of heart failure. These medications should be used in those who remain symptomatic after treatment with ACE or ARB, BB and diuretics. High levels of potassium (which has the potential to cause fatal heart rhythms) and low blood pressure are the most common side effects. Potassium deviations are less likely with eplerenone. While this drug is a diuretic and helps remove fluid its main effect is through the effect it has on the hormone aldosterone.

Digoxin

This was once the number one prescribed drug for heart failure but has fallen out of favor with recent medical research. Digoxin works by helping the heart beat more effectively. This drug has shown not to prolong life, but can be effective in reducing hospitalizations and helping the patient feel better, especially patients with systolic heart failure (4).

Digoxin can be very dangerous and needs to be monitored. Blood levels are drawn to assure they are in the therapeutic range, as fatal heart rhythms can develop when the level of this drug becomes toxic. Side effects include loss of appetite, nausea, vomiting, diarrhea and blurred vision.

Nitrates and Hydralazine

For years this combination has been used in heart failure management in those who do not tolerate ACE-I or ARB. Recently it was determined that when added to standard therapy, isosorbide dinitrate and hydralazine diminishing morbidity and mortality in

African-Americans with class III or IV heart failure.

The African American Heart Failure Trial (A-HeFT) demonstrated improvement in morbidity and mortality, including a 39% reduction in hospitalization, improvement in quality of life or functional status, and a 43% reduction in mortality (5).

Diastolic heart failure

Some heart failure patients have preserved left ventricular function. Data is lacking on effective management strategies for diastolic heart failure. Treatment involves treating any underlying heart conditions such as high blood pressure, coronary heart disease or any problems with the valves in the heart. Diuretics are often used in these patients because they are common afflicted with extra fluid. Treatment of high blood pressure is a cornerstone of treatment and can be accomplished with a variety of medications. Salt intake should be restricted.

Device therapy

Device therapy is often implemented after failure of medical therapy to adequately control the disease. Patients with heart failure are at high risk for experiencing heart rhythm disturbances that can lead to death. Treatment is accomplished with either pacemakers, internal cardiac defibrillators (ICD) or both.

Pacemakers are helpful because they help the two sides of the heart contract more efficiently together. Patients with heart failure often have conduction disturbances that cause the right and left side of the heart to not beat together effectively. Pacemakers help the heart beat more blood out of its left ventricle, reduces the amount of strain that is placed on the heart, and may even slow down the changes that occur in the heart caused by heart failure.

Patients with heart failure are at high risk for developing fatal arrhythmias. ICD shock the heart back into rhythm if they develop a fatal arrhythmias

Further studies are needed to determine the usefulness of these therapies.

Summary

Heart failure is a very serious disease, which will affect a larger portion of the population. While there is currently no cure, there are many effective treatments to improve the quality and quantity of life.

Reference:

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outcomes of patients with preserved systolic function hospitalized for heart failure: a report from the OPTIMIZE-HF Registry. Journal of the American College of Cardiology 2007; 50(8): 768-77.

Table 1 Causes and Contributing Factors of Heart Failure

- Chronic hypertension
- Myocardial infarction
- Valvular heart disease
- Cardiomyopathy
- Ischemic heart disease
- Obesity
- Diabetes
- Physical inactivity
- Excessive alcohol intake
- Cigarette smoking
- Chemotherapeutic agents
- Certain toxins (cobalt and lead)
- Hyperlipidemia
- Sleep apnea
- Abnormal thyroid
- Myocarditis
- Sarcoidosis
- Hemochromatosis

Table 2 Signs and Symptoms of Heart Failure

Symptoms

- Paroxysmal nocturnal dyspnea
- Shortness of breath
- Dyspnea on exertion
- Weight gain
- Cough
- Fatigue

Signs

- Crackles
- S3 gallop
- Bilateral ankle edema
- Hepatomegaly/ascites
- Increased heart rate
- Jugular venous distention
- Displaced apical impulse

