NATURAL MEDICINE IN TREATING HEART DISEASE

Inflammation and Colon Play Key Roles

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Natural and Alternative Medicine are now getting great recognition in the proper evaluation and treatment with many heart related conditions effecting Americans today. This brief article will give you, the reader, insight on many facets which are key components to how effective and successful treating heart disease can be with natural and alternative medicine.

It is estimated that some 68 million Americans suffer from some form of heart disease this year, and the numbers keep growing. Some 1 million Americans will have a heart attack this year.

As frightening as this number is, over 1 million more will die of some type of cardiovascular disease, with the leading killer being congestive heart failure. Cardiovascular disease is responsible for 41 percent of all deaths in the United States each year and half of all deaths worldwide.

Incredibly, cardiovascular disease kills more people than the next eight leading causes of death combines – and each year, these deaths exceed the total U.S. death rates in World War I and Ii combines.

Of the cardiovascular diseases, congestive heart failure affects some 5 million Americans and 10 million Europeans. While scientists cannot agree on the causation of heart failure, they know a number of conditions that can lead up to it, including repeated heart attacks, uncontrolled hypertension, certain toxins, chronic inflammation, progressive atherosclerosis, and complications of certain medications.

The truly frightening thing is that while the number of heart attacks has stabilized or even improved somewhat over the past decade, the incidence and death rates from heart failure have increased.

Recent studies have shown that people diagnosed with heart failure have a 50 percent to 65 percent chance of dying of their disease within five years of the diagnosis

Growing evidence indicates, however, that there doesn't have to be such a grim prognosis.

What is Heart Failure?

The heart is a hollow ball of muscle that normally is about the size of your fist. This incredible organ beats over 100m000 times a day, pumping some 2,000 gallons of blood through 60,000 miles of blood vessels – that's equal to almost two and a half times around the planet.

This four chambered pumping machine is divided into right and left systems. The right atria and ventricle pump blood to the lungs to pick up oxygen and get rid of carbon dioxide, and the left atria and ventricle pump this oxygenated blood to all the organs and tissues of your body.

In heart failure, one, and eventually both, of these pumping systems are defective, so that blood begins to back up in the veins, causing engorgement of poorly oxygenated blood in our tissues and organs.

This leads to a number of symptoms:

Shortness of breath especially with exertion Swelling of the ankles Chronic, sometimes bloody, cough Extreme weight loss Extreme fatigue

In the early stages, the heart tries to fight back by enlarging its muscles, like a bodybuilder. The harder is gets until finally the muscle gives out, causing the blood to back up in the lungs, resulting in massive leakage of fluid (plasma0 into the lungs.

Before the frightening endgame begins, these patients suffer from a number of organ failures and related problems.

This is because the blood that has backed up in the veins causes engorgement in all organs and tissues of the body, including the kidneys, lungs, gastrointestinal tract, and even the brain.

Worse, this blood has very low levels of oxygen (a condition called hypoxia), which deprives these organs and tissues of this vital factor.

What Newer Studies Are Finding

Heart failure, like so many other conditions, is strongly linked to chronic inflammation and specific immune over activity.

The debate among scientists is simply this: Which comes first? Is the inflammatory immune reaction the cause of the heart failure, or is the inflammation caused by the heart failure and, once started, makes it progress rapidly?

So far, we only know for sure that it causes the condition to advance – fast.

Studies of many heart failure patients have clearly shown that the levels of certain inflammation markers, such as the high-sensitivity C-reactive protein (hsCRP), interleukin-6 (IL-6) and tumor necrosis factor-alpha TNF-, accurately predict who will have the most rapidly advancing heart failure and who is more likely to die.

Likewise, high levels of inflammatory cytokines, such as TNF-, IL-6, and CRP, may in f act cause a number of the symptoms of heart failure.

It was first reported in 1990m that blood levels of TNF- were much higher in people with heart failure than people with normal heats – some 10 times higher. Those with the highest levels of TNF- had the most advanced disease and had the worst weight loss (called cachexia).

Further evidence was found when researchers examined diseased hearts that had been removed during heart transplantation and found that these heart hearts cold produce the TNF-themselves and that it did not come just from blood.

In another study, doctors followed 152 heart-failure patients for at least 12 months. Researchers found that measures of inflammatory markers. (TNF-, s TNFR2, IL_6, and CD14) accurately predicted, independently, who would have the poorest prognosis.

Of these factors, the more powerful predictor was the TNF-level). Those with the highest levels of TNF- (sTNFR1) had a 12-fold higher risk of mortality than those with the lowest levels of TNF-.

Yet another study found that risk was highly correlated to hsCRP blood levels.

What this means is that the more intense the inflammation, the greater the damage to the heat and the more aggressively the heart muscle will degenerate.

This finding solves a quandary cardiologists faced for so many years: Why do some heart-failure patients seem to do fairly well while others deteriorate rapidly and die?

Inflammation, especially chronic elevation of heart muscle and blood levels of inflammatory cytokines, directly and indirectly, causes massive destruction of the heart:

Destroys heart-muscle cells

Damages the lining of coronary blood vessels and cardiac microvessels

(called endothelium)

Damages the heart's electrical conduction system

Inflammation results in very high levels of free radicals and lipid peroxidation products in the heart muscle, activation of prostaglandin pathways that further accelerate inflammation, and formulation of free radicals.

What all this means is that even if inflammation is not the primary cause of heart failure, it plays a major role in the acceleration and worsening of heart failure.

I believe that inflammation is a major player in heart failure itself. If we look at all the known links to heart failure, such as hypertension (high blood pressure), failure of heart valves, mercury exposure, infections and atherosclerosis – they are all associated with high levels of inflammatory cytokines.

So, no matter the actual initiating clinical event, it is chronic, low-level inflammation and the resulting high levels of oxidative stress that are causing the progressive destruction of the heart muscle.

While the inflammatory cytokine TNF- has gotten a lot of attention, both as a marker for heart failure prognosis and as a target for treatment, the inflammatory cytokine-IL-6 (interleukin-6) may be even more important.

Studies have shown that IL-6 levels are much higher in patients with the most severe heart failure and that it directly damages the heart muscle, leading to deterioration of heart-function lab tests, such as heart filling, pressure, ejection fractions, and cardiac output. In fact, cardiology researchers consider IL-6, to be a very strong independent risk factor—the higher the level, the greater the risk.

CRP is also a strong independent risk factor, and we know that it is IL-6, that drives the production of CRP in the liver. CRP is not only a marker for inflammation; it is a powerful inflammatory cytokine itself.

Role of the Gut in Heart Failure

Here is one of the mysteries of heart failure; What is the source of the continuous immune-inflammatory reactions? In 1997, Dr. Stefan D. Anker ad co-workers

proposed the hypothesis that, as the heart failure progresses, blood engorges and swells the lining of the colon and intestine, causing bacteria from the colon to move into the bloodstream (leaky gut syndrome).

This triggers an immune attack, raising inflammatory cytokine levels (TNF-, IL-6 and IL-1) and, because bacteria continue day by day to drift into the bloodstream, the problem only gets worse over time.

Subsequent studies confirm what they had suspected. In fact, other researchers gave a series of patients with severe heart failure antibiotics that killed the colon bacteria. They found a significant drop in patient TNF- levels, as well as other inflammatory markers. High levels of TNF- are inflammatory markers known to cause extreme wasting and muscle loss, as seen in severe cases of heart failure.

The colon contains more than 400 species of organisms. Repairing the damage to the gut and replacing the beneficial bacteria (probiotics) makes even better sense.

Inflammatory cytokine buildup in the blood and heart triggers enormous concentrations of destructive free radicals and lipid-peroxidation products. If there are not effectively removed, they will deplete the heart's antioxidant defenses—including its antioxidant enzymes (SOF, catalase and GSHPx).

Once the antioxidant defenses are depleted, the free radicals and lipid=peroxidation products become even more destructive. It's like removing the police in a riot—more things get destroyed.

This explains why some heart-failure patients begin to deteriorate very rapidly and why good nutrition plays such a vital role in heart protection.

Studies have shown that as the heart failure progresses, free radicals and lipid-peroxidation products begin to increase dramatically, not just in the heart, but in all organs that are being deprived of oxygen.

Ironically most cardiologists do nothing to correct this. Rather, they just add new drugs.

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