Obesity and Cardiomyopathy

The link between obesity and insulin resistance and atherosclerosis has been well established in modern literature. Researchers have recognized the relationship between obesity driven low-grade inflammation and cytokine (cellular secretions) dysfunction and alteration. These pathophysiological changes explain why metabolic and heart disease manifest rapidly in obese persons, particularly when the level of visceral fat dominates or heavily contributes to adiposity. Obesity-related changes in cytokine concentration are known to increase risk for diabetes and atherosclerosis. Interestingly, further connections between the pro-inflammatory secretion of cellular chemicals and premature disease and death have been made related to heart dysfunction. Clinical trials analyzing the relationship between obesity and cardiomyopathy have demonstrated direct association between visceral fat levels and changes in heart function in otherwise healthy normotensive obese persons.

Obesity-related low-grade inflammation is identifiable by changes in chemical indicators associated with the inflammatory process including C-reactive protein (CRP), interleukin-6 (IL-6), and tumor necrosis factor-alpha (TNF). These measurable inflammatory chemicals are elevated in obese individuals with the most dramatic increases in persons with central girth measures above 40 inches. When normotensive obese subjects were placed on echocardiograms (ECG) these same pro-inflammatory chemicals correlated with both echocardiographic abnormalities and the level of central adiposity. Elevated levels of visceral fat predispose obese persons to premature cardiac dysfunction by increasing circulating cytokines and their soluble receptors. In study, subject exposure to these chemicals, void of blood pressure instigation, experienced morphological and functional alterations to the heart tissue, including end-diastolic septum thickness, end diastolic wall thickness, absolute and indexed left ventricular mass, and decreased heart performance indexes.

Complementary studies have duplicated these outcomes with and without the presence of metabolic disease. In all cases obese individuals show cytokine dysfunction leading to changes in echocardiographic parameters with the negative impact proportional to the level of visceral fat storage. Interestingly, although some of the normotensive, obese subjects had type II diabetes and impaired glucose tolerance, it was the level of central adiposity that predicted the most important morphological and functional echocardiographic alterations. End-diastolic septum and posterior wall thickness and the left ventricular mass were significantly greater in subjects with a waist circumference >40 inches.
With or without the presence of metabolic disease, waist circumference, waist-to-hip ratio, and gender were statistically associated with echocardiographic alterations whereas other disease characteristics were not.

Further evidence links these same cytokine disturbances with ventricular dyssynchrony and changes in heart contraction velocities. Likewise, an association between insulin resistance and left ventricular overload and consequent increases in ventricular mass has been established. Both may help explain the fact that obesity is a well-established risk factor for congestive heart failure. Congestive heart failure occurs when the heart tissue is modified and heart valve function is compromised, dramatically reducing the efficiency of the heart. Independently, dyssynchronous ventricular contraction and volume overload associate with increased susceptibility to congestive heart failure. Together the effects may increase the probability of developing life-threatening heart dysfunction. In both cases cytokine variations were identified consistent with the low-grade inflammation caused by obesity.

It seems apparent that obesity, particularly associated with high visceral fat content, has deleterious effects on cardiac tissue and function. This further supports the importance of reducing levels of body fat in obese individuals with and without the presence of metabolic disease and hypertension. Weight loss of at least 10% of body weight has shown to yield positive effects upon both cytokine secretion and ventricular function including ventricular dyssynchrony. Following 1 year of multidisciplinary intervention women who lost at least 10% of body weight experienced a reduction in pro-inflammatory cytokine concentrations and improvements in echocardiographic parameters.

Physical activity has also demonstrated a positive effect on cytokine and receptor activity. Following four weeks of physical activity improvements in cytokine expression were evident. Even a single bout of exercise showed positive enhancement to muscle cell receptor and enzyme activity. These findings support diet and exercise strategies both for weight loss and improved tissue function to reduce or prevent the consequences of obesity related inflammation. Preventing obesity before it occurs is still the best method for avoiding the onset of disease and physiological changes that lead to premature disease and death.