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Effects of Exercise and Losing Weight: Review

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Abstract

Both obesity and atrial fibrillation (AF), which both contribute to an increase in the incidence of cardiovascular disease events, are reaching epidemic proportions. Cardiovascular hemodynamics and cardiac structure and function are negatively impacted by obesity, as is the prevalence of AF, which is partly linked to electroanatomical remodeling in obese patients. However, a number of studies, including those on AF, have shown that overweight and obese patients with these disorders have a better prognosis than thinner patients with the same severity of cardiovascular disease and AF. The authors of this paper talk about special issues with AF in obese patients as well as the evidence that, despite the obesity paradox, weight loss, increased cardiorespiratory fitness, and physical activity/exercise training can improve the prognosis of obese patients with AF.

Keywords: Weight loss; Obesity; Diet

Introduction

Experts now refer to this epidemiological trend as an AF epidemic because its prevalence is expected to nearly triple over the next three decades, despite the fact that AF is already regarded as the most common sustained arrhythmia in adults [1]. By 2030, the estimated prevalence in the United States will reach 12.1 million, up from the current 5.2 million. Although many things contribute to this AF epidemic, obesity, which causes left atrial (LA) remodeling through a variety of mechanisms, is linked to a significant increase in the risk of developing AF. However, similar to other cardiovascular diseases, there is evidence that despite the fact that obesity increases the risk of AF, overweight and obese patients with AF appear to have a better prognosis, including CVD and all-cause survival, than lean AF patients. This suggests an apparent obesity paradox [2].

Numerous known cardiovascular conditions have been independently linked to the onset of AF in early epidemiological studies. Obesity has recently emerged as a distinct risk factor for AF. Obesity, for instance, has been identified as a significant, modifiable risk factor for AF by long-term prospective data from the Framingham Heart Study with nearly 14 years of follow-up. Importantly, obstructive sleep apnea, a common comorbid condition in obese individuals, has not been found to be associated with obesity and AF [3]. In addition, the Women's Health Study data have elegantly demonstrated the dynamic nature of the AF risk associated with changes in weight. In particular, short-term weight gain to a BMI >25 kg/m^2 was found to increase the risk of AF, and obese people who lost weight to a BMI 30 kg/m^2 over five years had a lower risk of AF than those who maintained a BMI 30 kg/m^2 over the same time. Given that obesity is the second-highest population-attributable risk for AF after hypertension, it is likely to increase the global burden of AF in the coming decades. Genetically predicted obesity and AF incidence are linked, according to a recent large study, suggesting the need for early prevention in the AF epidemic [4].

Literature Review

A number of hemodynamic changes that predispose to changes in cardiac morphology, which may lead to ventricular dysfunction, are linked to obesity. Patients with mild-to-moderate obesity may experience fewer of these changes, but those with severely obese patients may experience more of them. The total and central blood volumes rise when there is an excess of adipose tissue and more lean body mass [5]. The majority of obese people experience an increase in cardiac output as a result of these changes, which are facilitated by a decrease in systemic vascular resistance (SVR). The rise in cardiac output is primarily due to an increase in left ventricular (LV) stroke volume because there is little change in heart rate. Excessive cardiac output increases the risk of LV hypertrophy and enlargement (LVH). Recent research suggests that elevated cardiac output and decreased SVR are not always linked to central obesity. In these patients, LV remodeling is more likely to be concentric than eccentric. In obese patients with abnormal LV geometry, there is growing evidence that concentric LV remodeling and hypertrophy occur as frequently or more frequently than eccentric LVH. The development of LVH may be influenced by comorbidities like obesity-related systemic, neurohormonal, and metabolic changes (activation of the renin-angiotensin-aldosterone system and sympathetic nervous systems, hyperinsulinemia due to insulin resistance, hyperleptinemia due to leptin resistance, and possibly lipotoxicity). LV diastolic dysfunction may occur if the wall stress on the LV increases sufficiently [6]. LV systolic dysfunction may accompany LV diastolic dysfunction if LV wall stress is not keeping up with LVH. In many cases, LV failure as a result of LVH and the associated adverse LV loading conditions causes an increase in pulmonary venous pressure, pulmonary capillary pressure, and left lung volume. In severe obesity, pulmonary arterial hypertension is common. It is mostly caused by HF on the left side, but sleep apnea and obesity hypoventilation, both common in severe obesity, may make it easier. Right atrial enlargement, RV failure, and right ventricular (RV) enlargement and hypertrophy are all possible outcomes of pulmonary hypertension in conjunction with elevated cardiac output. The term "obesity cardiomyopathy" refers to heart failure that is entirely or primarily caused by severe obesity [7,8].

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Discussion

The central hemodynamic changes caused by excessive adipose accumulation and their subsequent effects on cardiac morphology and ventricular function in severely obese patients. In severe obesity, left ventricular (LV) hypertrophy can be eccentric or concentric. Obesity severity and duration, adverse LV loading conditions (particularly hypertension) duration and severity, and possibly neurohormonal and metabolic abnormalities like increased sympathetic nervous system tone, activation of the renin-angiotensin-aldosterone system, insulin resistance with hyperinsulinemia, leptin resistance with hyperleptinemia, adiponectin deficiency, lipotoxicity and lipoapoptosis are all factors that influence LV remodeling and geometry. The development of LV failure may be aided by these modifications. Right ventricular (RV) failure may follow LV failure, which is facilitated by sleep apnea/obesity hypoventilation-related pulmonary arterial hypertension [8].

Conclusion

The risk of AF is significantly raised in the context of obesity and weight gain because of the close connection between these disorders. However, there is evidence to suggest that higher CRF levels appear to be associated with the primary prevention of AF, and weight-loss programs, as well as programs that increase physical activity, exercise, and CRF levels, reduce recurrences of AF in patients who have a history of the condition. The effects of CRET and other weight-loss programs, as well as physical activity and exercise, on the prevention of other major cardiovascular disease (CVD) events, such as coronary heart disease (CHD) and heart failure (HF), as well as CVD- and all-cause mortality in patients with AF, require further investigation.

Acknowledgement

None

Conflict of Interest

None

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