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A Missed Preventive Opportunity: Severe Obesity Progressing to Obstructive Sleep Apnea, Atrial Fibrillation and Advanced Heart Failure

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Introduction:

Severe obesity is a well-established risk factor for obstructive sleep apnea (OSA), atrial fibrillation (AF), and heart failure, yet obesity-directed therapy is frequently delayed or overlooked. Early intervention may alter disease trajectory and prevent progression to advanced cardiomyopathy.

Case Presentation:

A 44-year-old unemployed, wheelchair-bound male with severe obesity (BMI 51 kg/m²; weight 153 kg; height 171 cm) and multiple cardiometabolic comorbidities, including hypertension, dyslipidaemia, gout, and hyperthyroidism, was diagnosed in 2014 with non-ischemic dilated cardiomyopathy and a severely reduced left ventricular ejection fraction of 18%. Transthoracic echocardiography demonstrated severe biventricular and bi-atrial dilatation with moderate mitral regurgitation. The patient previously declined left ventricular assist device implantation and listing for heart transplantation. He underwent cardiac resynchronization therapy with defibrillator (CRT-D) implantation in December 2023. His clinical course was complicated by persistent atrial fibrillation resulting in recurrent episodes of acute decompensated heart failure, requiring atrioventricular node ablation in April 2024. Pro BNP levels improved from 28,000 pg/mL pre-procedure to 8,000 pg/mL post-ablation. Despite optimized device therapy and guideline-directed medical therapy, he continued to experience multiple hospital admissions for heart failure decompensation. Notably, he had never received pharmacological or surgical treatment for obesity. Due to recurrent admissions, he was referred to a cardiovascular risk reduction clinic to address severe obesity as a potentially modifiable driver of his advanced heart failure. On assessment, he expressed strong motivation for weight loss. Given high clinical suspicion of untreated OSA contributing to refractory heart failure, he was referred for sleep assessment and polysomnography with planned initiation of continuous positive airway pressure therapy. Initial financial barriers limited access to obesity care; however, charitable funding has since enabled further management.

Conclusion:

This case highlights severe obesity and suspected OSA as potentially reversible contributors to advanced heart failure in a relatively young patient. Delayed obesity management represents a missed preventive opportunity likely contributing to the development of OSA, AF, and progressive cardiomyopathy. Obesity should be addressed as a central disease driver rather than a comorbidity in heart failure management.