One case of severe obesity with preserved ejection fraction heart failure treated by permanent cardiac pacemaker

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December 1, 2020

Abstract

A 54 year old male, with multiple diseases(such as severe obesity, severe edema, atrial fibrillation, high atrioventricular block, global enlargement (lA55mm LV 62mm RA 69*55mm RV 46mm)), was diagnosed as heart failure with preserved ejection fraction (HFpEF). After treatment with drugs and implanting permanent cardiac pacemaker, the patient's edema completely disappeared, weight loss of 42kg, heart failure symptoms disappeared left ventricular shrinkage (LV 59mm).

Title Page

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Running title: Cardiac pacemaker implantation in a patient with severe obesity and heart failure

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Abstract

A 54 year old male, with multiple diseases(such as severe obesity, severe edema, atrial fibrillation, high atrioventricular block, global enlargement (lA55mm LV 62mm RA 69*55mm RV 46mm)), was diagnosed as heart failure with preserved ejection fraction (HFpEF). After treatment with drugs and implanting permanent cardiac pacemaker, the patient's edema completely disappeared, weight loss of 42kg, heart failure symptoms disappeared left ventricular shrinkage (LV 59mm).

Conflict of Interest : None

Text

We provide one case with clinical features: 1. A 54-year-old male with severe obesity. 2. The patient had progressive edema and wheezing for half a year. 3. Atrial fibrillation history for half a year; overweight since childhood; left lower limb fracture with osteomyelitis for 3 years. 4. Physical examination: end sitting position, asthmatic appearance, bilateral lung breathing sounds thick, bilateral lower lung bottom respiratory sounds low, no dry and wet rales. The heart rate was 45 beats / min, and the heart sound was low and blunt. No pathological murmur and extra heart sound were found in the auscultation area of each valve. Abdominal distention, pigmentation, tough skin texture, local subcutaneous induration, percussion dullness. 5. ECG: atrial fibrillation, high degree of atrioventricular block, bradycardia (ventricular rate 36 beats / min); UCG: cardiac enlargement (LA55mm LV 62mm RA 69 * 55mm RV 46mm), LVEF 50%, left ventricular filling dysfunction. 6. Surgeons suspected that the edema was caused by lymphatic reflux disorder. We consider his diagnosis as ejection fraction preserved heart failure(HFpEF). However, what is the etiology and pathogenesis, what is the mechanism, and how should we deal with it?

At present, overweight and obesity have become a global "epidemic". According to WHO, 39% of the world's population over the age of 18 is overweight, of which 13% are obese. In China, obesity accounts for one ninth of China's total population. Moreover, a large number of studies have shown that obesity is closely related to cardiovascular disease. According to the Framingham Heart Study, an increase in BMI of $1\text{kg}/\text{m}^2$ increases the risk of heart failure by 5% in men and 7% in women. Studies[1] on heart failure(HF) show that 32% - 49% of HF patients are obese; after 20 years of obesity, the incidence of HF increases by 70%. Therefore, combined with the individual characteristics of the patients, we speculate that obesity leads to the structural and electrical remodeling of the cardiac cavity, and the slow ventricular rate caused by atrial fibrillation and high atrioventricular block leads to the occurrence of HFpEF. But, what is the mechanism of HFpEF caused by obesity?

Obesity leads to heart failure through both direct and indirect mechanisms[1,2]. The direct mechanism includes: 1. Hemodynamic changes: BMI increased $5\text{kg}/\text{m}^2$ as systolic blood pressure increased by 5mmHg. 2. Activate renin angiotensin aldosterone system: promote interstitial cardiac fibrosis, platelet aggregation and endothelial dysfunction, and finally lead to left ventricular hypertrophy(first to diastolic and then systolic dysfunction). 3. Promote the expression of inflammatory cytokines (TNF - α, IL-6, etc.): cause myocardial fibrosis, increase myocardial stiffness, and finally lead to left ventricular hypertrophy. 4. Obesity cardiomyopathy: the accumulation of cardiac lipid leads to myocardial thickening, early enhancement of myocardial contractility and increase of cardiac output to adapt to excessive body weight oxygen consumption; at the same time, free fatty acid induced myocardial lipid toxicity can lead to apoptosis of cells with high lipid content (such as myocardial cells), causing myocardial fibrosis, leading to the decrease of myocardial contractility. Indirect factors include: 1. Insulin resistance increases the risk of diabetes, coronary heart disease and cerebrovascular disease, forms a vicious cycle with weight gain. 2. Atrial fibrillation and atrioventricular block induce heart failure, even sudden death; electrical remodeling caused by myocardial structural remodeling, such as myocardial infiltration, adipocytes and fibrosis when the atrium is involved, leads to heterogeneous atrial pulse conduction. Fibrosis will replace normal tissues, when atrioventricular node, bundle branch and other structures are involved, result in the generation, spread and transmission of electrical signals, causing atrioventricular block and bundle branch block.

The above mechanism can explain the case we reported. Therefore, we speculate that the disease of HFpEF in this patient is due to obesity, and its pathogenesis is as follows: in the early stage of obesity, myocardial structural remodeling formed leads to compensatory expansion of heart and increase of cardiac output and decrease of cardiac diastolic function; in the middle stage of obesity, atrial fibrillation and high degree of atrioventricular block caused by myocardial electrical remodeling occurs, which further decreases the diastolic function of heart, reduces the amount of return heart blood, decreases the cardiac output dsignificantly, eventually leads to systemic and pulmonary circulation congestion, results in heart failure symptoms. Fortunately, the patient has not yet progressed to an advanced stage of decreased myocardial contractility. Now, the pathogenesis and process is clear. But, how to treat?

The non cardiovascular comorbidities of HFpEF are diverse and the pathological mechanism is complex.

There is no evidence-based medical scheme to choose from [3]. Therefore, in accordance with the principles of disease treatment of symptomatic treatment and eliminating incentives and treating etiology and coping with complications and guiding prognosis, we have developed a four-step strategy: 1. Diuretics were used to improve symptoms. 2. A permanent pacemaker is put in to solve the problem of slow ventricular rate, and the golden Triangle (angiotensin II receptor antagonist + beta blocker + aldosterone receptor antagonist) were used to delay cardiac remodeling(LV: 62mm VS 59mm). 3.The coronary artery CTA was performed to confirm the coronary artery lesions, and lipid-lowering therapy was added after the coronary artery atherosclerosis was confirmed; Arterial and venous ultrasound and CTA of both lower extremities were performed to determine the lymphatic reflux and arteriovenous conditions of the lower extremity, and elastic stockings were recommended when the venous valve regurgitation was serious;5.Prognosis management, such as diet control + exercise weight loss. Then, there is a question, the main cause of the patient is obesity, why did we not put "weight loss" in the first place? That is because: 1. Patients with improved cardiac function can tolerate various measures of weight loss (exercise, diet control, surgical gastric volume reduction, etc.); 2. "Obesity paradox": obesity not only increases the risk of cardiovascular abnormalities, but aslo has a good prognosis in developed diseases. This phenomenon[1,2] has been observed, including acute and obesity Chronic heart failure, coronary heart disease, acute myocardial infarction, hypertension and atrial fibrillation. Therefore, we put weight loss into the step of prognostic management, and developed a non-surgical weight loss plan in respect of the patient's wishes, with good results (weight loss of 7kg in 3 months). In addition, we did not do coronary artery related examination first considering for rationality. It is more reasonable to choose cardiac pacemaker first when the patients who need both pacemaker implantation and coronary stent implantation, because of the safety of antiplatelet therapy and contrast induced nephropathy.

Finally, we suggest : 1. Weight control should be started from childhood, and the public should be educated about the relationship between obesity and cardiovascular disease to prevent the occurrence of such diseases; 2. It is suggested that pacemakers should be implanted in time when conditions permit, so as to reduce the occurrence and development of sudden death and heart failure.

Reference

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3. Upadhya B, Kitzman DW. Heart failure with preserved ejection fraction: New approaches to diagnosis and management. Clin Cardiol. 2020 Feb;43(2):145-155. Fig.1 Weight change Fig.2 ECG change

	waist circumference	hip circumference	right thigh	left femur	right fibula	left fibula	right ankle	left ankle
Before treatment（cm）	156.2	190	74	70	59	67	42	49
After treatment（cm）	136	165	63	62	44	49	22	38