Clinical characteristics of dilated cardiomyopathy patients with different body weight statuses and the impact of weight management on patient prognosis: a preprint

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Abstract

Background Obesity is closely associated with the occurrence and progression of dilated cardiomyopathy (DCM), but the differences in clinical characteristics among DCM patients with different body weight status and the prognostic value of weight management remain unclear. Objective To investigate the baseline clinical characteristics of DCM patients with different body weight status and to analyze the impact of weight management on their prognosis. Methods This single-center prospective cohort study categorized patients according to BMI into a normal-weight group (BMI <24 kg/m^2), an overweight group (24 kg/m^2 ≤ BMI <28 kg/m^2), and an obese group (BMI $ 28kg/m^{2}). Baseline clinical data were collected, and patients were followed up for 12 months after discharge via outpatient visits or telephone to record the occurrence of major adverse cardiovascular events (MACE). According to the magnitude of 12-month weight change, patients were divided into three groups: weight change <5%, 5% ≤ weight change <10%, and weight change $ 10 $^2 = 16.83, P <0.001). Univariate Cox proportional hazards regression analysis showed that follow-up BNP, LVEF, cardiac function at follow-up, magnitude of weight change, and the use of GLP-1 receptor agonists, mineralocorticoid receptor antagonists (MRA), and sodium–glucose cotransporter 2 inhibitors (SGLT2i) were independent factors influencing the occurrence of MACE in DCM patients (all P <0.05). After adjusting for sex, diabetes, smoking history, drinking history, and medication use, multivariate Cox proportional hazards regression analysis with MACE as the dependent variable and magnitude of weight change as the independent variable demonstrated that the magnitude of weight change was independently associated with the occurrence of MACE in DCM patients (P <0.05). Subgroup analysis showed that a greater magnitude of weight change was associated with

a reduced risk of MACE (HRoverall = 0.89, 95% CI = 0.81–0.98, P = 0.018). Interaction analysis indicated that across subgroups stratified by sex, age, presence of diabetes, and use of SGLT2i, MRA, or GLP-1 receptor agonists, the association between greater magnitude of weight change and reduced MACE risk in DCM patients was consistent (P for interaction >0.05), all demonstrating a protective effect. However, the relationship between the magnitude of weight change and MACE risk differed significantly according to the use of β-blockers (P for interaction = 0.004). Conclusion DCM patients with BMI $ $24kg/m\hat{}\{2\}$$ are younger and have a higher prevalence of metabolic disorders such as hypertension and diabetes. After 12 months of weight management, patients with a weight reduction $ $10% exhibit the most pronounced improvement in cardiac function, manifested by a significant decrease in follow-up BNP, a marked increase in follow-up LVEF, and the lowest incidence of major adverse cardiovascular events. Therefore, it is recommended that structured weight management targeting a $ $10% weight reduction be incorporated into the comprehensive treatment of overweight/obese DCM patients to improve their cardiac function and clinical prognosis.

Full Text

Analysis of Clinical Characteristics in Dilated Cardiomyopathy Patients with Different Weight Statuses and the Influence of Weight Management on Prognosis

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Abstract

Background: Obesity is closely related to the occurrence and development of dilated cardiomyopathy (DCM). However, differences in clinical characteristics among DCM patients with distinct weight statuses and the prognostic value of weight management remain unclear.

Objective: To explore the baseline clinical characteristics of DCM patients with different weight statuses and to analyze the impact of weight management on their prognosis.

Methods: This single-center prospective cohort study enrolled DCM patients who were divided into normal weight (BMI<24 kg/m^2), overweight (24 kg/m^2 $\leq$ BMI<28 kg/m^2), and obese (BMI $\geq$ 28 kg/m^2) groups based on body mass index (BMI). Baseline clinical data were collected, and patients were followed up for 12 months through outpatient visits or telephone calls to record major adverse cardiovascular events (MACE). According to the magnitude of weight change during the 12-month period, patients were stratified into <5% weight change, 5%–10% weight change, and $ $10% weight change groups. Kaplan-Meier survival curves were plotted for MACE and survival time across the three groups. Univariate and multivariate Cox proportional hazards regression models and subgroup analyses were performed to evaluate the effect of weight management on MACE risk in DCM patients.

Results: A total of 322 obese patients with DCM admitted to the Affiliated Hospital of Jiangsu University from January 2022 to June 2024 were prospectively enrolled. Based on baseline BMI, patients were divided into normal weight (84 cases), overweight (132 cases), and obese (106 cases) groups. Significant differences were observed among the three groups in age, systolic blood pressure, diastolic blood pressure, left ventricular end-systolic diameter (LVSd), prevalence of hypertension, diabetes, coronary atherosclerosis, smoking history, and usage of soluble guanylate cyclase (sGC) agonists, orlistat, and glucagon-like peptide-1 (GLP-1) receptor agonists (all P<0.05). Among the three weight change groups, significant differences were found in admission weight, follow-up B-type natriuretic peptide (BNP), follow-up left ventricular ejection fraction (LVEF), follow-up cardiac function, MACE incidence, and GLP-1 receptor agonist usage (P<0.05). The magnitude of weight change during 12-month follow-up showed linear correlations with follow-up BNP (r=-0.158, P=0.004) and LVEF (r=0.229, P<0.001). Kaplan-Meier survival curves demonstrated significant differences in MACE incidence among the three weight change groups (χ^2=16.83, P<0.001). Univariate Cox regression analysis identified follow-up BNP, LVEF, follow-up cardiac function, weight change magnitude, and use of GLP-1 receptor agonists, mineralocorticoid receptor antagonists (MRA), and sodium-glucose cotransporter 2 inhibitors (SGLT2i) as independent influencing factors for MACE in DCM patients (P<0.05). After adjusting for gender, diabetes, smoking history, alcohol consumption, and medication use, multivariate Cox regression showed that weight change magnitude was independently associated with MACE occurrence in DCM patients (P<0.05). Subgroup analysis revealed that increased weight change was associated with reduced MACE risk (overall HR=0.89, 95%CI=0.81–0.98, P=0.018). Interaction analysis showed consistent protective effects of increased weight change across subgroups stratified by gender, age, diabetes status, and use of SGLT2i, MRA, or GLP-1 receptor agonists (P-interaction>0.05). However, the relationship between weight change and MACE risk differed significantly between patients with and without β-blocker use (P-interaction=0.004).

Conclusion: DCM patients with BMI $\geq$ 24 kg/m^2 are younger and have higher rates of metabolic disorders such as hypertension and diabetes. After 12 months

of weight management, patients with $ $10% weight loss showed the most significant cardiac function improvement, manifested by substantially decreased follow-up BNP, increased LVEF, and the lowest MACE incidence. Structured weight management targeting $ $10% weight loss should be integrated into the comprehensive treatment of overweight/obese DCM patients to improve cardiac function and clinical prognosis.

Keywords: Overweight; Obesity; Body weight changes; Weight management; Obesity paradox; Dilated cardiomyopathy; Prognosis

Introduction

Obesity has become a global public health problem, with WHO data indicating that over 650 million adults worldwide suffer from obesity. The pathogenesis of obesity is complex, involving genetic, environmental, behavioral, and psychological factors, with high-calorie diets, sedentary lifestyles, and insufficient sleep being the main environmental drivers. Obesity not only significantly increases the risk of metabolic diseases such as type 2 diabetes and hypertension but is also closely associated with various cardiovascular diseases. Studies have shown that obesity can lead to structural and functional cardiac changes, including left ventricular hypertrophy, diastolic dysfunction, and cardiac dilation. Additionally, pro-inflammatory cytokines secreted by adipose tissue (such as leptin and resistin) may cause insulin resistance and chronic low-grade inflammation, further exacerbating the burden on the cardiovascular system. Obesity affects both patients' quality of life and imposes a heavy economic burden on healthcare costs.

Dilated cardiomyopathy (DCM) is a myocardial disease characterized by left ventricular or biventricular dilation and systolic dysfunction. Its etiology is complex and diverse, including genetic factors, metabolic disorders, viral infections, alcohol abuse, and autoimmune diseases. DCM is primarily characterized by heart failure, arrhythmias, and increased risk of sudden death, with a 5-year survival rate of only 59%. Recent research has revealed a close relationship between obesity and DCM. Obesity can promote the development of DCM through multiple mechanisms, including chronic inflammatory states, insulin resistance, increased oxidative stress, and myocardial lipotoxicity. Studies have shown that adipose tissue in obese patients over-secretes pro-inflammatory factors such as tumor necrosis factor-α (TNF-α) and interleukin-6 (IL-6), which can cause cardiomyocyte apoptosis and fibrosis, thereby aggravating ventricular remodeling and dysfunction. Obese patients have 2.5 times higher risk of developing DCM than non-obese individuals, highlighting the importance of weight management in preventing cardiovascular complications in obese patients.

The complex relationship between obesity and cardiovascular disease has drawn widespread attention to the "obesity paradox," which describes how, in some chronic heart failure patients, overweight or mildly obese individuals have better

prognoses than those with normal or low weight. However, the universality and underlying mechanisms of this paradox in specific DCM populations remain unclear. Meanwhile, extreme obesity may also exacerbate myocardial remodeling through hemodynamic load and metabolic abnormalities, leading to deterioration of cardiac function.

Therefore, this study aims to investigate the differences in clinical characteristics, echocardiographic parameters, and biomarker expression among DCM patients with different weight statuses, and to prospectively evaluate the impact of structured weight management interventions on cardiac function improvement, rehospitalization rates, and all-cause mortality, thereby providing more optimized treatment strategies for clinical practice.

Methods

Study Design and Population

This single-center prospective cohort study prospectively enrolled 322 obese patients with DCM admitted to the Affiliated Hospital of Jiangsu University from January 2022 to June 2024.

Inclusion criteria: (1) Met diagnostic criteria for obesity according to the "Guidelines for Long-term Weight Management and Clinical Application of Drugs in Obese Patients (2024 Edition)" and DCM criteria according to the "Chinese Guidelines for Comprehensive Management of Cardiomyopathy 2025" and New York Heart Association (NYHA) functional classification I-IV; (2) Standardized use of cardiomyopathy medications; (3) Age >18 years, willing to lose weight, clear consciousness, complete clinical data, good compliance; (4) Signed informed consent.

Exclusion criteria: (1) Combined with other cardiomyopathies or coronary atherosclerotic heart disease; (2) Use of immunosuppressants or chemotherapy within the past six months; (3) History of eating disorders, endocrine diseases affecting weight (such as thyroid dysfunction), use of weight-affecting drugs (such as glucocorticoids), pregnancy or lactation; (4) Patients who underwent cardiac resynchronization therapy (CRT), CRT-defibrillator (CRT-D), cardiac contractility modulation (CCM), or left ventricular assist device implantation due to worsening cardiac function during follow-up; (5) Patients with severe infectious diseases.

The study was approved by the Medical Ethics Committee of the Affiliated Hospital of Jiangsu University (KY2025K0906).

Data Collection

General clinical data were collected for obese patients with DCM, including demographic information (age, sex, BMI, smoking history, alcohol consumption), clinical characteristics (NYHA functional class, blood pressure, heart

rate), comorbidities (hypertension, diabetes, atrial fibrillation, chronic kidney disease), laboratory indicators [cardiac troponin I (cTnI), B-type natriuretic peptide (BNP)], and echocardiographic parameters [left ventricular end-diastolic dimension (LVDd), left ventricular end-systolic diameter (LVSd), left ventricular ejection fraction (LVEF)].

All patients received guideline-recommended pharmacological therapy for heart failure, with medications titrated to maximum tolerated doses over 2-4 weeks based on patient tolerance. Treatment continued for 12 months with follow-up data collection. Concurrently, patients underwent dietary control, physical exercise, cardiac rehabilitation training, and some received orlistat or glucagon-like peptide-1 (GLP-1) receptor agonists (liraglutide or semaglutide) under endocrinology consultation.

Grouping by BMI

Patients were divided into three groups based on BMI: normal weight (BMI<24 kg/m^2), overweight (24 kg/m^2 $\leq$ BMI<28 kg/m^2), and obese (BMI $\geq$ 28 kg/m^2).

Follow-up and Endpoints

Patients were followed up at 1, 3, 6, and 12 months after discharge through outpatient visits or telephone calls for a total of 12 months. Follow-up content included BNP, LVEF, cardiac function, weight change magnitude, and usage of GLP-1 receptor agonists, mineralocorticoid receptor antagonists (MRA), sodium-glucose cotransporter 2 inhibitors (SGLT2i), and soluble guanylate cyclase (sGC) agonists.

The primary endpoint was major adverse cardiovascular events (MACE), including: (1) sudden cardiac death, (2) rehospitalization due to worsening heart failure symptoms, (3) arrhythmia or thromboembolic events, (4) heart transplantation or left ventricular assist device implantation.

Weight Monitoring

All enrolled patients underwent 12 months of multidisciplinary weight management. Weight was measured using the same model of smart scale (precision $±$0.1 kg) every Wednesday morning after overnight fasting, with data automatically synchronized to a dedicated research application. The intervention effect was calculated as: Weight change (%) = [(12-month follow-up weight - baseline weight) / baseline weight] × 100%, with baseline weight measured at enrollment.

Weight change groups were defined as: <5% weight change, 5%–10% weight change, and $ $10% weight change.

Statistical Analysis

Statistical analysis was performed using SPSS 25.0 software. Normally distributed continuous variables were expressed as mean ± standard deviation and compared using one-way ANOVA. Non-normally distributed continuous variables were expressed as median (P25, P75) and compared using Kruskal-Wallis H test. Categorical variables were expressed as percentages and compared using χ^2 test. Spearman rank correlation was used to assess the relationship between weight change magnitude and follow-up BNP/LVEF. Kaplan-Meier analysis evaluated MACE occurrence across the three weight control groups. Univariate and multivariate Cox proportional hazards regression models were used to analyze MACE influencing factors. Subgroup and interaction analyses were performed to explore the robustness and consistency of the association between weight change magnitude and MACE risk. Two-sided tests were performed with $P<0.05$ considered statistically significant.

Results

Baseline Characteristics by BMI Groups

Based on BMI, patients were divided into normal weight (84 cases), overweight (132 cases), and obese (106 cases) groups. No significant differences were observed among the three groups in gender, heart rate, admission cTnI, admission BNP, LVDd, LVEF, atrial fibrillation, chronic kidney disease, alcohol consumption, family history of heart disease, NYHA functional class distribution, or usage rates of ACEI/ARB/ARNI, β-blockers, SGLT2i, and MRA ($P>0.05$). However, significant differences were found in age, systolic blood pressure, diastolic blood pressure, LVSd, prevalence of hypertension, diabetes, coronary atherosclerosis, smoking history, and usage of sGC agonists, orlistat, and GLP-1 receptor agonists ($P<0.05$).

Pairwise comparisons showed that the obese group was younger than the other two groups, and the overweight group was younger than the normal weight group ($P<0.05$). The obese group had higher usage rates of orlistat and GLP-1 receptor agonists than the other groups, with the overweight group higher than the normal weight group ($P<0.05$). Systolic and diastolic blood pressure, as well as the prevalence of hypertension, diabetes, and smoking history, were higher in both the overweight and obese groups compared to the normal weight group ($P<0.05$). The obese group had lower LVDd, LVSd, and coronary atherosclerosis prevalence than the normal weight group, and lower sGC agonist usage than the overweight group ($P<0.05$) .

Comparison of Patients with Different Weight Change Magnitudes at 12 Months

Based on weight change magnitude after 12 months of weight management, patients were divided into <5% weight change (115 cases), 5%–10% weight

change (157 cases), and $ $10% weight change (50 cases) groups. No significant differences were found in 12-month follow-up weight or orlistat usage among the three groups (P>0.05). However, significant differences were observed in admission weight, follow-up BNP, follow-up LVEF, follow-up cardiac function distribution, MACE incidence, and GLP-1 agonist usage (P<0.05). Specifically, the $ $10% weight change group had higher admission weight and follow-up LVEF, but lower follow-up BNP, MACE incidence, and GLP-1 agonist usage compared to the other two groups (P<0.05). The 5%–10% weight change group had lower MACE incidence than the <5% weight change group (P<0.05) .

Correlation Between Weight Change Magnitude and Follow-up BNP/LVEF

Spearman rank correlation analysis revealed that 12-month weight change magnitude was linearly correlated with follow-up BNP (r=-0.158, P=0.004) and LVEF (r=0.229, P<0.001) [Figure 1: see original paper].

Prognostic Comparison Across Different Weight Change Magnitudes

Kaplan-Meier survival curves showed significant differences in MACE incidence among the three weight change groups (χ^2=16.83, P<0.001) [Figure 2: see original paper].

Univariate Cox regression analysis identified follow-up BNP, LVEF, follow-up cardiac function, weight change magnitude, and use of GLP-1 receptor agonists, MRA, and SGLT2i as independent influencing factors for MACE in DCM patients (P<0.05) .

After adjusting for gender, diabetes, smoking history, alcohol consumption, medication use, and age, multivariate Cox regression analysis showed that weight change magnitude remained an independent influencing factor for MACE in DCM patients (P<0.05) .

Subgroup analysis demonstrated that after adjusting for multiple covariates, increased weight change magnitude was associated with reduced MACE risk (overall HR=0.89, 95%CI=0.81–0.98, P=0.018). Interaction analysis showed consistent protective effects of increased weight change across subgroups stratified by gender, age, diabetes status, and use of SGLT2i, MRA, or GLP-1 receptor agonists (P-interaction>0.05). However, the relationship between weight change and MACE risk differed significantly between patients with and without β-blocker use (P-interaction=0.004), with the protective effect of weight loss being pronounced in the β-blocker subgroup [Figure 3: see original paper].

Discussion

This study systematically evaluated the clinical value of weight management in DCM patients with different weight statuses. Overweight and obese DCM patients showed a younger age trend and higher prevalence of hypertension and

diabetes. After 12 months of weight management, the magnitude of weight change was significantly correlated with improvements in cardiac function indicators (decreased BNP, increased LVEF). Furthermore, weight management was an independent protective factor for reducing MACE risk after multivariate adjustment, and this association remained consistent across most clinical subgroups. These findings collectively support the important clinical significance of integrating proactive weight management into standard DCM treatment strategies.

Based on our results and existing literature evidence, systematic weight control in overweight/obese DCM patients is necessary for multiple reasons. Obesity-induced metabolic abnormalities (such as insulin resistance and lipotoxicity) and hemodynamic changes (such as increased blood volume and cardiac load) can directly accelerate DCM progression. Clinical evidence indicates that overweight and obesity are not only independent cardiovascular risk factors but also important direct causes of myocardial structural and functional changes. The improvement in cardiac function (such as increased LVEF and decreased BNP) and clinical prognosis benefits from weight management may be related to the reversal of multiple pathophysiological changes associated with overweight/obesity. First, chronic low-grade inflammation triggered by adipose tissue accumulation (characterized by elevated pro-inflammatory factors such as TNF-α and IL-6) can directly damage cardiomyocytes and promote fibrosis. Second, obesity-related insulin resistance and hyperinsulinemia are key drivers accelerating myocardial fibrosis and ventricular remodeling. Weight management may directly reduce the toxic effects of these processes on the myocardium by improving insulin sensitivity. Therefore, structured weight management interventions simultaneously target these core inflammatory and metabolic mechanisms, thereby significantly improving cardiac function indicators and prognosis in overweight/obese DCM patients.

Our results showed that the $ $10% weight change group had higher admission weight and follow-up LVEF but lower follow-up BNP, MACE incidence, and GLP-1 receptor agonist usage compared to other groups. The 5%–10% weight change group had lower MACE incidence than the <5% weight change group, suggesting that weight control can improve cardiac remodeling indicators in overweight/obese DCM patients and may enhance the efficacy of conventional heart failure medications. Some studies have also recommended incorporating weight control into the comprehensive management of these patients as an important supplement to pharmacotherapy. Clinical practice should emphasize multidisciplinary collaboration to develop personalized and sustainable weight intervention strategies to optimize long-term prognosis in DCM patients.

The 12-month weight change magnitude showed linear correlations with follow-up BNP (r=-0.158, P=0.004) and LVEF (r=0.229, P<0.001). Univariate Cox regression showed that follow-up BNP, LVEF, cardiac function, weight change magnitude, and use of GLP-1 receptor agonists, MRA, and SGLT2i were independently associated with MACE (P<0.05). Multivariate Cox regression after

adjusting for gender, diabetes, smoking/alcohol history, and medication use confirmed that weight change magnitude was independently associated with MACE ($P<0.05$). Subgroup analysis showed that after adjusting for multiple covariates, increased weight change magnitude remained a protective factor against MACE events. These findings suggest that evaluating treatment response through weight change magnitude is crucial for adjusting heart failure medications, guiding defibrillator implantation, and reducing MACE events.

Our study findings appear to contrast with the widely reported "obesity paradox" phenomenon in heart failure, where higher BMI is associated with lower mortality in chronic, stable heart failure populations. However, it is important to recognize that our study differs fundamentally from observational studies supporting the "paradox." The latter describe a static association potentially confounded by factors such as muscle reserve and nutritional status, whereas our study focuses on dynamically changing weight status through active intervention. Our results suggest that actively reducing obesity may provide additional benefits beyond static BMI associations by improving metabolic disorders and reducing cardiac load. Thus, our study offers an important complementary perspective to the "obesity paradox": weight management should not be neglected in heart failure patients with obesity simply because of their higher BMI. The significant benefits from weight management may involve multi-level synergistic mechanisms. At the hemodynamic level, weight loss directly reduces systemic blood volume and peripheral vascular resistance, thereby decreasing cardiac preload and afterload and creating favorable conditions for reverse remodeling. At the metabolic level, weight loss effectively reverses myocardial lipotoxicity—the accumulation of lipid intermediates in cardiomyocytes when excessive free fatty acids exceed oxidative capacity, leading to insulin resistance, mitochondrial dysfunction, and apoptosis. Improved insulin resistance enables cardiomyocytes to utilize glucose more efficiently, optimizing energy metabolism. Additionally, adipose tissue (especially visceral fat) is a major source of pro-inflammatory cytokines (such as TNF-α and IL-6) that directly impair myocardial contractility and promote fibrosis. Successful weight management has been shown to significantly reduce circulating levels of these cytokines, thereby attenuating their sustained attack on the myocardium. In summary, the benefits of weight management result from the combined effects of macroscopic load reduction and improvements in microscopic lipotoxicity, insulin resistance, and specific inflammatory pathways.

Limitations

This single-center clinical observation has several limitations: relatively small sample size, short follow-up duration, heterogeneity in weight intervention measures, and lack of randomization. Some subgroups had small sample sizes ($n<20$), resulting in unstable point estimates and confidence intervals for hazard ratios. These results should be considered exploratory and interpreted cautiously. Future studies should adopt multicenter randomized controlled de-

signs with longer follow-up periods and explore the differential effects of various weight intervention strategies (such as dietary patterns, exercise prescriptions, and pharmacological assistance). Future directions include exploring biomarker-guided personalized weight loss strategies, the impact of bariatric surgery on advanced DCM patients, and artificial intelligence-assisted remote weight management models. Advanced imaging techniques (such as cardiac magnetic resonance) to assess myocardial tissue characteristic changes may help elucidate the mechanisms of weight control.

In conclusion, active and structured weight management, particularly achieving $ $10% weight loss, may have important clinical value in improving cardiac function and reducing cardiovascular event risk in overweight/obese DCM patients.

References

[1] CHE Xingxing, GAO Ting, HOU Qingbin, et al. Effect of weight management on cardiac function in patients with chronic heart failure with reduced ejection fraction[J]. Chinese Journal of Integrative Medicine on Cardio-/Cerebrovascular Disease, 2020, 18(3): 483-487. DOI:10.12102/j.issn.1672-1349.2020.03.026.

[2] SONG S Y, TIEN C L, CUI H, et al. Myocardial rev-erb-mediated diurnal metabolic rhythm and obesity paradox[J]. Circulation, 2022, 145(6): 448-464. DOI:10.1161/CIRCULATIONAHA.121.056076.

[3] XIE Yinghao, LÜ Deliang, PENG Ke, et al. Interaction analysis of overweight, obesity and hypertension on stage B heart failure[J]. Chinese Journal of Prevention and Control of Chronic Diseases, 2024, 32(4): 260-263, 268. DOI:10.16386/j.cjpccd.issn.1004-6194.2024.04.005.

[4] MUKHTAR G, SASIDHARAN B, KRISHNAMOORTHY K M, et al. Clinical profile and outcomes of childhood dilated cardiomyopathy-A single-center three-decade experience[J]. Ann Pediatr Cardiol, 2023, 16(3): 175-181. DOI: 10.4103/apc.apc_{{{149}}_{{22}}}.

[5] AN Xuanqi, ZHANG Jian, ZHOU Qiong. Clinical research review: Impact of obesity on the development and outcome of heart failure[J]. Chinese Journal of Heart Failure and Cardiomyopathy, 2020, 4(3): 222-230. DOI: 10.3760/cma.j.cn101460-20191120-00087.

[6] SMART C D, FEHRENBACH D J, WASSENAAR J W, et al. Immune profiling of murine cardiac leukocytes identifies triggering receptor expressed on myeloid cells 2 as a novel mediator of hypertensive heart failure[J]. Cardiovasc Res, 2023, 119(13): 2312-2328. DOI: 10.1093/cvr/cvad093.

[7] ANTONOPOULOS A S, PANAGIOTOPOULOS I, TERENTES-PRINTZIOS D, et al. Relationship between obesity and hypertrophic or dilated cardiomyopathy: The role of sex[J]. Clin Obes, 2024, 14(6): e12692. DOI: 10.1111/cob.12692.

[8] IBÁÑEZ B, VILLENA-GUTIERREZ R. Cardiac mitochondrial transplantation: the force awakens[J]. J Am Coll Cardiol, 2021, 77(8): 1089-1092. DOI:10.1016/j.jacc.2021.01.017.

[9] Chinese Society of Endocrinology. Guidelines for long-term weight management and clinical application of drugs in obese patients (2024 edition)[J]. Chinese Journal of Endocrinology and Metabolism, 2024, 40(7): 545-564. DOI: 10.3760/cma.j.cn311282-20240412-00149.

[10] National Cardiovascular Center Cardiomyopathy Alliance, Cardiovascular Precision Medicine Branch of China International Exchange and Promotive Association for Medical and Health Care, SONG Lei, et al. Chinese guidelines for comprehensive management of cardiomyopathy 2025[J]. Chinese Circulation Journal, 2025, 40(5): 420-462. DOI: 10.3969/j.issn.1000-3614.2025.05.002.

[11] AKBARI T, HAMMERSLEY D J, MAY C Y, et al. The impact of cardio-renal-metabolic profile in dilated cardiomyopathy[J]. Curr Cardiol Rep, 2025, 27(1): 89. DOI: 10.1007/s11886-025-02241-8.

[12] RAYNER J J, ABDESSELAM I, D' ARCY J, et al. Obesity-related ventricular remodelling is exacerbated in dilated and hypertrophic cardiomyopathy[J]. Cardiovasc Diagn Ther, 2020, 10(3): 559-567. DOI: 10.21037/cdt-19-587.

[13] PACKER M, ZILE M R, KRAMER C M, et al. Tirzepatide for heart failure with preserved ejection fraction and obesity[J]. N Engl J Med, 2025, 392(5): 427-437. DOI: 10.1056/NEJMoa2410027.

[14] RAYNER J J, PETERZAN M A, CLARKE W T, et al. Obesity modifies the energetic phenotype of dilated cardiomyopathy[J]. Eur Heart J, 2021, 43(9): 868-877. DOI: 10.1093/eurheartj/ehab663.

[15] BUTLER J, SHAH S J, PETRIE M C, et al. Semaglutide versus placebo in people with obesity-related heart failure with preserved ejection fraction: a pooled analysis of the STEP-HFpEF and STEP-HFpEF DM randomised trials[J]. Lancet, 2024, 403(10437): 1635-1648. DOI: 10.1016/S0140-6736(24)00469-0.

[16] JIA Xiaoyan, LIAN Ruijia, MA Baodong, et al. Effect of laparoscopic sleeve gastrectomy on cardiac structure and function in obese patients with heart failure[J]. Acta Academiae Medicinae Sinicae, 2025, 47(2): 226-236. DOI: 10.3881/j.issn.1000-503X.16179.

[17] PARK J, SONG H, MOON S, et al. Cardiometabolic benefits of fenofibrate in heart failure related to obesity and diabetes[J]. Cardiovasc Diabetol, 2024, 23(1): 343. DOI: 10.1186/s12933-024-03489-4.

[18] WANG X H, LIU W, LI H L, et al. Exploring the role of obesity in dilated cardiomyopathy based on bio-informatics analysis[J]. J Cardiovasc Dev Dis, 2022, 9(12): 462. DOI: 10.3390/jcdd9120462.

[19] SOLOMON S D, OSTROMINSKI J W, WANG X W, et al. Effect

of semaglutide on cardiac structure and function in patients with obesity-related heart failure[J]. J Am Coll Cardiol, 2024, 84(17): 1587-1602. DOI: 10.1016/j.jacc.2024.08.021.

[20] VEST A R, SCHAUER P R, RODGERS J E, et al. Obesity and weight loss strategies for patients with heart failure[J]. JACC Heart Fail, 2024, 12(9): 1509-1527. DOI: 10.1016/j.jchf.2024.06.006.

[21] ALEBNA P L, MEHTA A, YEHYA A, et al. Update on obesity, the obesity paradox, and obesity management in heart failure[J]. Prog Cardiovasc Dis, 2024, 82: 34-42. DOI: 10.1016/j.pcad.2024.01.003.

[22] TUTOR A W, LAVIE C J, KACHUR S, et al. Updates on obesity and the obesity paradox in cardiovascular diseases[J]. Prog Cardiovasc Dis, 2023, 78: 2-10. DOI: 10.1016/j.pcad.2022.11.013.

[23] BUTT J H, PETRIE M C, JHUND P S, et al. Anthropometric measures and adverse outcomes in heart failure with reduced ejection fraction: revisiting the obesity paradox[J]. Eur Heart J, 2023, 44(13): 1136-1153. DOI: 10.1093/eurheartj/ehad083.

[24] WU M X, TAN J, CAO Z Y, et al. Sirt5 improves cardiomyocytes fatty acid metabolism and ameliorates cardiac lipotoxicity in diabetic cardiomyopathy via CPT2 de-succinylation[J]. Redox Biol, 2024, 73: 103184. DOI: 10.1016/j.redox.2024.103184.

[25] GOSWAMI S K, RANJAN P, DUTTA R K, et al. Management of inflammation in cardiovascular diseases[J]. Pharmacol Res, 2021, 173: 105912. DOI: 10.1016/j.phrs.2021.105912.

Author Contributions: SUN Xia and ZHONG Wei conceived the study and wrote the manuscript; SHEN Wen and TANG Xiang designed the study protocol; WANG Kailin monitored weight changes and performed data visualization; DAI Zhiyin, ZHANG Chaopu, and YUAN Wei standardized the format and performed data analysis; YUAN Guoyue revised the final manuscript and takes responsibility for the paper.

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Note: Figure translations are in progress. See original paper for figures.