European Heart Journal Supplements (2004) **6** (Supplement F), F87—F90

# Obesity, weight loss and heart failure

**Rita Mariotti***, **Francesca Castrogiovanni, Francesco Becherini, Bernardo Cortese, Lorenzo Rondinini, Mario Mariani**

*Cardiac and Thoracic Department, University of Pisa, via Paradisa 2, 56124 Pisa, Italy*

**KEYWORDS**
Obesity;
Heart failure;
Tailored diet

**Background** The current scientific data controversially indicate obesity both as a risk factor for developing congestive heart failure (CHF) and a positive prognostic factor.
**Aims** The present study evaluated the impact of weight loss on clinical and instrumental parameters in a selected group of obese patients with CHF.
**Methods** An overall population of 560 HF patients was sub-grouped on the basis of the Body Mass Index (BMI): 8.2% were underweight (BMI < 20.7), 63.9% normal weight (BMI: 20.7—27.7), 19.6% overweight (BMI: 27.8—31), and 8.3% obese (BMI > 31). Of the 46 overweight and obese patients, 28 (55.2% men, age 51—80 years) accepted a tailored low-caloric dietary program for at least 4 months. The 28 patients belonged to both obese and overweight groups (BMI > 27.8) and were in NYHA classes II—III. Mean follow-up was 5 months.
**Results** The mean loss of body weight was 4 kg in 81.4% of patients, versus 3 kg mean increase in whole (560 patients) population (72.5—75.5 kg). In the 28 patients we recorded a significant ($p < 0.05$) improvement of NYHA class, better control of arterial blood pressure and statistically significant ($p < 0.05$) lowering of total cholesterol and triglyceride levels.
**Conclusions** Tailored dietetic program may improve clinical and instrumental parameters in patients with CHF.


## Introduction

The role of obesity in congestive heart failure (CHF) is widely debated. Recent data from Framingham Study show an increased risk of developing CHF in subjects with elevated body mass index (BMI) (5% in men and 7% in women for every increasing point of BMI).[1] However, some Authors underline the controversial role of obesity as an independent risk factor,[2] while others indicate obesity as a positive prognostic factor. On these bases, there are doubts about encouraging weight loss in obese patients with optimally treated CHF.[3]

Obesity directly and indirectly promotes diabetes mellitus II (DM), hypertension (HT) and dyslipidemia. These latter are leading risk factors for coronary artery disease, and ultimately for CHF.[1,4] Therefore, obesity should be treated as other risk factors, in patients with and without CHF. However, the correlation between obesity with prognosis in patients with CHF is not proven. Horwich et al.[3] examined the relationship between obesity and mortality in a population of 1203 patients with advanced CHF. They found that an elevated BMI was not a risk factor for mortality and was even associated with a better prognosis. This result, recently confirmed by Lissin et al.[6] in 522 patients, has been further validated by Gruberg et al.[7] for coronary artery disease (CAD). They evaluated the role of obesity in patients who had previously undergone PTCA, and found that patients with BMI < 18.5 and patients with BMI between 18.5 and 24.5 had a higher risk of mortality and in-hospital complications, with a risk of mortality 1 year higher than patients with BMI > 25.

* *Correspondence:* Tel.: +39 50 996799; fax: +39 50 577239.
*E-mail address:* r.mariotti@dcap.med.unipi.it (R. Mariotti).

1520-765X/$ - see front matter 
doi:10.1016/j.ehjsup.2004.09.013

With the aim of investigating the real impact of a therapeutic approach integrating medical therapy optimisation, tailored dietary program and correct lifestyle in obese patients with CHF, we performed a pilot dietary project at the Heart Failure Unit of Cardio-Thoracic Department of University of Pisa.

## Materials and methods

A population of 560 HF patients, enrolled during the first year of the research program into the experimental project of Heart Failure Unit, were sub-grouped on the basis of the BMI, according to Horwich et al.[3] 8.2% Patients were underweight (BMI < 20.7), 63.9% normal weight (BMI: 20.7—27.7), 19.6% overweight (BMI: 27.8—31), 8.3% obese (BMI > 31) (Fig. 1). Among the overall population of 46 overweight and obese patients, 28 (57% men, age 51—80 years): agreed to follow a tailored dietary program for at least 4 months. The ethiology of the CHF was: 27% CAD, 25% HT, 32% CAD + HT, 12% Idiopathic Cardiomyopathy, 4% Valvular Heart Disease. All these patients were in stable conditions and in optimised medical therapy. Mean doses of therapeutic agents were: digoxin 0.125 mg/die, furosemide 23.3 mg/die, carvedilol 21.1 mg/die, spironolactone 48.4 mg/die. The evaluation of the mean doses of ACE-inhibitors (in 24 patients, enalapril in 16) and AT-II-blockers (in 4 patients), assumed by all the patients, was prevented.

The clinical program included the contribution of a dedicated nutritionist who worked in collaboration with cardiologists and whose program of patient information, education, diet administration, and controls was additional to that performed by cardiologists and integrated in the overall clinical management plan.

These patients underwent a tailored low-calorie diet addressed to their clinical and metabolic conditions with the aim of reducing 500 kcal daily intake. The diet set up was: 55—60% carbohydrates, 15% proteins, 25—30% fats. A parallel dietary educational program was run to address the self-awareness of patient's alimentary errors and to improve their eating habits. We administered simple information about:

- the major alimentary substance groups;
- the differential role of dietary fats;
- the need of limiting sodium intake, water intake and alcohol abuse;
- the role of potassium intake;
- the benefits of abolishing smoking and carrying out physical activity.

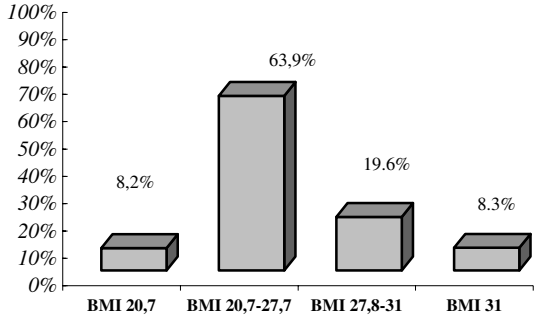


**Fig. 1** BMI representation in 560 HF patients.

Patients underwent medical and instrumental controls and tailored changes in their dietary programs. Each patient filled in a multiple-choice questionnaire to test his specific and general knowledge/cultural background, prior dietary programs, if any, and prior information about weight-loss.

At the patient's enrolment in the dietary program, anthropometric, history, and physical data were recorded. These data included family history of obesity and CHF, BMI, family history for cardiovascular risk factors, NYHA class, a modified congestion score as derived by Lucas et al.,[7,8] rhythm and heart rate, blood pressure and drugs therapy.

The congestion score[9] was modified to separate symptoms (dyspnoea and orthopnoea) from objective signs. Each of the following signs were scored one point each: third heart sound, hepatomegaly, jugular distension, leg oedema and pulmonary rales (maximum score 5). Subsequent follow-up occasions included recording of all modifiable parameters.

At the first and last control we performed a complete colour Doppler echocardiographic examination to compare left ventricular volumes and ejection fraction (EF %), and collected the biochemistry data, including glycemia, lipid profile (total, HDL and LDL cholesterol, tryglicerides) and renal function indexes (urea, creatinine, uric acid).

## Statistical methods

We calculated the mean values and standard deviations of the selected clinical and biochemical data as well as of the echocardiographic measurements. Results in the follow-up were compared with the *t*-test for coupled data using the NCSS 2001-PASS 2000 program.

## Results

### Whole population setting

The mean weight loss in the study population was 4 kg in 81.4% of patients, versus a mean increase of 3 kg in the 560 patients (72.5—75.5 kg).

### Compliance to the dietary restriction

Of the 28 patients, 23 positively complied to their tailored dietary program, while (Y) 5 did not respect the dietary indications (non-compliant patients).

### Weight loss in the compliant population

A mean weight loss of 4 kg has been recorded between enrolment and end of the follow-up in 81.4% of the 28 patients (from 89 to 85 medium weight, $p < 0.05$). The remaining 18.6% of the obese patients did not show any weight reduction, due to the above lack of compliance to the dietary restriction. During the same period the general population had a mean weight increase of 3 kg (from 72.5 to 75.5 kg).

**Table 1** Therapeutic doses in first and last controls

| Drugs | First dietetic evaluation | | Last control | | *p* |
|---|---|---|---|---|---|
| | Mean doses | Patients | Mean doses | Patients | |
| Furosemide | 23.3 mg/die | 19 | 24.01 mg/die | 20 | NS |
| Carvedilol | 21.09 mg/die | 14 | 21.61 mg/die | 13 | NS |
| Digoxin | 0.125 mg/die | 13 | 0.125 mg/die | 13 | NS |
| Spironolactone | 51 mg/die | 12 | 49.2 mg/die | 12 | NS |

### Congestion score

From the first dietary control to the last check-up the congestion score did not statistically modify (from 1.08 to 1.0; p. NS). Medical therapy was unmodified (Table 1).

### Clinical and biochemical findings

NYHA functional class improved from an average of 2.3—2.0 ($p < 0.05$). Total blood cholesterol and thryglyceride levels decreased from 190 to 178 mg/dl ($p < 0.05$), and from 158 to 123 mg/dl ($p < 0.05$), respectively. EF (from 39.9% to 40.5%), glycemia (from 127 to 125 mg/dl) and creatinine (from 1.1 to 1.0 mg/dl) did not show statistically significant changes.

### Statistical correlation

The weight loss was directly correlated with the number of clinical controls, both cardiological and nutritional, in female patients, and inversely correlated with the general cultural level. Body weight at enrolment did not correlate with the final weight loss (Table 2).

## Discussion

The present results support the hypothesis of a beneficial clinical contribution of weight loss in obese and overweight patients with CHF. Although the series is small, the data indicate that in this subset of patients the strategy of integration of optimal medical treatment with dietary education and tailored programs could provide a feasible, low-cost contribution to the overall management of the CHF syndrome. Our data need validation in larger populations of obese patients enrolled with identical criteria and treated with identical protocols.

**Table 2** Relationship between weight loss and cultural level, sex and number of controls

| | Patients with weight loss *N* = 23 (%) | Patients without weight loss *N* = 5 (%) |
|---|---|---|
| *Cultural level* | | |
| Low | 79 | 58 |
| High | 21 | 42 |
| *Sex* | | |
| Males | 47 | 86 |
| Females | 53 | 14 |
| *Number of controls* | | |
| 2 | 29 | 70 |
| >2 | 71 | 30 |

The non marginal prevalence of obese patients in the CHF patient population[3,5,10] indicates that an estimated relevant number of cases could benefit from dietary-based weight loss.

The discrepant data reported in the current literature on obesity as a prognostic factor in CHF should be carefully analysed in order to understand the relative weight of cachectic patients in the reported series. The obese and overweight patients should be investigated as a per se population, before and after dietary restriction and weight loss as well as integration between the optimisation of medical treatment with the benefits that dietary control of salt intake, for example, adds to the optimisation of medical treatment. Therefore, available data are not addressing the true problem, whose correct focus is tailored dietary programs and weight loss in obese patients with CHF rather than the role of weight loss in prognostic stratification of the whole CHF population. This approach to the analysis of the problem could also be beneficial for cachectic patients. The distinction between cachectic and non-(cachecting) cachectic patients is essential before reaching the conclusion that prognosis in CHF is better in obese and overweight patients than in patients with normal body weight.[3,10,11] Lavie hypothesises that lower body weight is associated with a heightened metabolic state and, in turn, with higher levels of tumour necrosis factor and other cytokines, shaping the ''cardiac cachexia'' of severe HF.[12] The globalisation of CHF programs, both treatments and types of study, is progressively masking specific needs and peculiarities of sub-groups of patients in whom low-cost interventions could modify prognosis or simply improve the quality of life. Dietary programs imply an educational impact. The beneficial strength of communication with patients can positively increase with appropriate levels of information and with an increase in the amount of time dedicated to the patients.

For instance the intestinal absorption in patients with CHF caused by CHD could be impaired by a concomitant atherosclerotic and ischemic disease affecting splancnic district: this is a topic totally neglected by current literature and medical attention. The overall problem of co-morbidity, especially in old patients, should be

considered in future research and the set up of clinical support strategies. Future studies dedicated to the body composition in CHF using the Dual Energy X-ray Absorptiometry could help clinicians to understand specific needs in nutritional support versus absorption and metabolism resources. This is unmet by current knowledge in cachectic patients.

Although the relationship between a strict dietary program and weight loss seems to be confirmed in both hypertensive and non-hypertensive obese patients,[13–17] there is a further problem in the current literature dedicated to CHF and obesity: the combination of obesity and hypertension. The benefits of dietary restriction for obesity may contribute to controlling salt intake and positively influence the results. Data should be independently evaluated in obese non-hypertensive and obese hypertensive patients in order to measure the effects of dietary education and regulation and weight loss, salt intake, as well as lowering of circulating lipids and glycemia. Age could additionally influence data, as young hypertensive and obese patients show extremely positive results of weight reduction on the left ventricular mass.[15,17,18]

Data from the present series show a statistically significant reduction of blood lipids: it is not yet clear if a modification of risk profile for CAD is effective in the CHF outcome. An Italian trial (GISSI-Heart Failure) has been designed to answer this question. Given the correlation between diabetes and obesity we expected an improved glycemic control that vice-versa did not reach statistical significance in our series. This failure could be due to the low series, or to the limited weight loss, that may not be enough to significantly decrease the insulin-resistance of obese patients. Available data in the current literature refer to obese patients without CHF, in whom the haemodynamic state has not deteriorated and the need for medication is low.

Finally, with regard to known effects of diet therapy in CHF our data indicate a better response in women and inverse correlation with cultural level. The instrumental data improved without treatment variations.

## Conclusions

Present data underline the need for dedicated clinical studies that integrate optimal medical treatments with tailored dietary programs in larger populations of obese and overweight patients. Any clinical improvement that can be reached without the administration of additional substances/drugs is a positive contribution in the management of patients with CHF. Furthermore, the educational effect of a dietary program and strict follow-up may positively reflect on the quality of life. The additional human resources required for this type of programs include cardiologists and nutritionists whose contribution extends from obesity, cachexia, and optimisation of the compliance to therapy.

## References


1. Kenchaiah S, Evans JC, Levy D et al. Obesity and the risk of heart failure. *New Engl J Med* 2002;**347**:305–13.
2. Wilson PWF, D'Agostino RB, Levy D et al. Prediction of coronary heart disease using risk factor categories. *Circulation* 1998;**97**:1837–47.
3. Horwich TB, Fonarow GC, Hamilton MA et al. The relationship between obesity and mortality in patients with heart failure. *J Am Coll Cardiol* 2001;**38**:789–95.
4. Yusuf S, Pitt B. A lifetime of prevention, the case of HF. *Circulation* 2002;**106**:2997–8.
5. Wilson PW, Kannel WB. Obesity, diabetes, and risk of cardiovascular disease in the elderly. *Am J Geriatr Cardiol* 2002;**11**:119–23.. 125.
6. Lissin LW, Gauri AJ, Froelicher VF et al. The prognostic value of body mass index and standard exercise testing in male Veterans with congestive heart failure. *J Card Fail* 2002;**8**:206–15.
7. Gruberg L, Weissman NJ, Waksman R et al. The impact of obesity on the short-term and long-term outcomes after percutaneous coronary intervention: the obesity paradox? *J Am Coll Cardiol* 2002;**39**: 578–84.
8. Lucas C, Johnson W, Hamilton MA et al. Freedom from congestion predicts good survival despite previous class IV symptoms of heart failure. *Am Heart J* 2002;**140**:840–7.
9. Cody RJ. The need for a sodium retention score in clinical trials of heart failure. *Clin Pharmacol Ther* 1993;**54**:7–10.
10. Massie BM. Obesity and heart failure – risk factor or mechanism?. *New Engl J Med* 2002;**347**:358–9.
11. Pi-Sunyer FX. Short-term medical benefit and adverse effect of weigth loss. *Ann Intern Med* 1993;**119**:722–6.
12. Lavie CJ, Milani R, Mehra MR et al. Obesity, weight reduction and survival in heart failure. *J Am Coll Cardiol* 2002;**39**:1563.. Discussion 1563–4.
13. Kanasky Jr WF, Anton SD, Rodrigue JR et al. Impact of body weight on long-term survival after lung transplantation. *Chest* 2002;**121**:401–6.
14. Davos CH, Doehner W, Rauchhaus M et al. Obesity and survival in chronic heart failure. *Circulation* 2000;**102**:II 876. (Abstract).
15. Himeno E, Nishino K, Nakashima Y et al. Weight reduction regresses left ventricular mass regardless of blood pressure level in obese subject. *Am Heart J* 1996;**131**:313–9.
16. Reisin E, Frolich ED, Messerli FH et al. Cardiovascular changes after weight reduction in obesity hypertension. *Ann Intern Med* 1983;**98**(March):315–9.
17. MacMahon SW, Wilcken DE, Mac Donald GJ. The effect of weight reduction on left ventricular mass. A randomised controlled trial in young, overweight hypertensive patients. *New Engl J Med* 1986;**314**:334–9.
18. Alpert MA, Terry BE, Kelly DL. Effect of weight loss on cardiac chamber size, wall thickness and left ventricular function in morbid obesity. *Am J Cardiol* 1985;**55**:783–6.