## **ORIGINAL ARTICLE**

# Exercise Tolerance Test Examination for Determination of Obesity Paradox in Patients with Systolic Heart Failure. A Clinical Comparative Study

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### ABSTRACT

Aims and objectives: Current study was conducted in Department of cardiology, Hayatabad medical complex Peshawar, for one year of duration. The aims and objective of this study was to deliver health awareness and description of a biological hypothesis i.e. obesity paradox, among our community.

Methodology: 250 patients with a history of cardiac complications were selected and divided into two different groups. 100 individuals with BMI > 25 were in group A and their history of systolic heart failure was positive while in Group-B 150 patients with BMI< 35 were selected and their systolic heart failure history were negative.

Conclusion: Obesity paradox provides survival against systolic heart failure in high BMI cardiac patients this was the findings of this study. On the other side cardiorespiratory fitness also play an important role in severity, and survival. It has seen that obesity was found protective in cardiac diseases and there is a strong relationship between body mass index and obesity paradox in heart failure. The standard mean deviation levels of BMI, systolic blood pressure, percentage of ECG variation, oxygen saturation of exercise tolerance test were significant (P<0.005) in group-B as compared to the group-A. Keywords: Obesity paradox, systolic heart failure, Body mass index, oxygen saturation

#### INTRODUCTION

Systolic heart failure, also known as left-ventricle heart failure. Systolic heart failure occurs when the heart does not pump effectively or contract properly between heartbeats [3]. Obesity is a well-known and major predictor of morbidity and mortality in patients with cardiovascular disease, and other illnesses [1]. Despite this, some studies show that obesity is linked to lower mortality in heart failure patients, a result known as the obesity paradox. Several possible explanations for the obesity paradox have been presented, albeit they are not fully understood [10]. Given the high frequency of obesity in patients with heart failure, understanding the obesity paradox has crucial clinical implications.

Obesity, as measured by a higher BMI, has been demonstrated to be protective against heart failure, leading to the widely accepted concept of an obesity paradox [9]. This perplexing evidence has been examined and investigated from several angles in a diverse group of heart failure patients, but it remains largely unexplained, despite conflicting and complimentary viewpoints being provided [5]. Low levels of adiponectin and a reduced response to sympathetic activation have been linked to a protective background, while elevated levels of serum lipoproteins have been linked to a protective background by counteracting bacterial cytokines and endotoxins [8]. The many interactions between obesity and important clinical factors of the natural history of the disease are currently under investigation, regardless of the protective neuro-hormonal and inflammatory function that an elevated BMI may play [9].

Confounding variables, according to some researchers, may explain part of the obesity conundrum. In both healthy individuals and cohorts with cardiovascular disorders, cardiorespiratory fitness is highly linked to prognosis [2]. Exercise capacity and other cardiopulmonary exercise test factors have been shown to be important in predicting prognosis in heart failure in a number of studies [4]. indeed, the 14ml o2/kg/min cut-off point for peak oxygen demand is still routinely used to classify patients with heart failure into low-risk and high-risk categories. Obesity paradoxes have also been observed in groups of people with coronary heart disease, but not in those who have high exercise tolerance [11]. Recently, similar results in systolic heart failure patients were reported [14]. Using a multicenter database based on

cardiopulmonary exercise test, we wanted to explore how exercise tolerance and cardiorespiratory capacity influenced the obesity paradox in a larger population of systolic heart failure patients [15].

Exercise intolerance, defined as a decrease in the ability to conduct physical activities accompanied by substantial dyspnea and/or fatigue, is a symptom of chronic heart failure and is linked to a lower quality of life and a higher mortality rate [16]. although heart failure is thought to be caused by a reduction in cardiac output at rest and during physical effort (lower cardiac reserve), it is now well acknowledged that patients do not acquire the heart failure condition in a vacuum. The pathophysiological underpinnings of exercise intolerance in heart failure are multifaceted, including impairments in cardiac and pulmonary reserve, as well as decreased peripheral and respiratory skeletal muscle perfusion and/or function, all of which can contribute to the illness in different ways [18]. we present a comprehensive review of the pathophysiology of exercise and functional intolerance in patients with heart failure, including a discussion of the various modalities used to quantify it, as well as the role of comorbid conditions in reducing exercise and functional capacity, emphasising the fact that different causes may coexist and contribute to exercise intolerance in patients with heart failure in a variety of ways [2].

#### MATERIALS AND METHODS

Current clinical study was conducted at department of cardiology, Hayatabad medical complex Peshawar for one year. For current study 250 patients with a history of cardiac complications were selected and divided them into two different groups. In Group-A, 100 individuals with a history of systolic heart failure and their BMI > 25 was noted, while in Group-B 150 patients with BMI< 35 and without systolic heart failure history were selected. Clinical cardiac history and previously reports of all patients were recorded.

In exercise tolerance test the capacity of exercise of an individual, the ability of his endure exercise and maximum work done during treadmill exercise period was measured Then, physical examination, laboratory analyses, ECG, and transthoracic echocardiography were performed, Pulse oximetry method was used to measure the oxygen saturation of each individual before and after treadmill exercise test. The collected raw data presented

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Bio-statistically through version SPSS-20 by standard mean deviations of different parameters.

#### RESULTS

Group-A: n=100 individuals with a history of systolic heart failure, age= 40-60 years

Variables	Before treadmill	After treadmill	P<0.005
	exercise	exercise	
	Mean ± SD	Mean ± SD	
Oxygen saturation Levels	95.01±01.02	93.01± 01.02	0.000

Group-B: n=150 individuals with cardiac problems but no systolic heart failure history, age= 40-60 years

Variables	Before treadmill	After treadmill	P<0.005
	exercise	exercise	
	Mean ± SD	Mean ± SD	
Oxygen saturation	98.01±01.02	94.01±01.02	0.000
Levels			

Variables	Previous Data	Current Data	P<0.005
	History	Mean ± SD	
	Mean ± SD		
BMI	35.5 ± 1.10	35.9 ± 1.10	0.000
Systolic blood	150 ± 1.11	140 ± 1.11	0.000
Pressure			
ECG variation %	3.4 ± 2.10	2.5 ± 2.10	0.000
Exercise Tolerance	18. 2± 0.2	19. 3± 0.2	0.000
Test at stage 5 %			

Variables	Previous Data	Current Data	P<0.005
	History	Mean ± SD	
	Mean ± SD		
BMI	24.5 ± 1.10	23.5 ± 1.10	0.000
Systolic blood	160.1± 1.11	150.1± 1.11	0.000
Pressure			
ECG variation %	20.2± 2.10	18.2± 2.10	0.000
Exercise Tolerance	13. 1± 0.2	14. 1± 0.2	0.000
Test at stage 5 %			





In present study grouping patients according to BMI showed a significant (P<0.005) changes regarding oxygen saturation. The

average age of cardiac patients in both groups were 40-60 years. The standard mean deviation levels of BMI, systolic blood pressure, percentage of ECG variation and percentage of exercise tolerance test at fifth stage after 6 month in Group-A and Group-B were  $(23.5 \pm 1.10, 150.1 \pm 1.11, 18.2 \pm 2.10, 14.1 \pm 0.2)$ ,  $(35.9 \pm 1.10, 140 \pm 1.11, 2.5 \pm 2.10, 19.3 \pm 0.2)$  respectively. The oxygen saturation levels of each individuals of group-A and group-B after and before treadmill exercise were  $(95.01 \pm 01.02, 93.01 \pm 01.02)$  (98.01 $\pm$  01.02, 94.01 $\pm$  01.02) were noted. The findings of this study stated that higher BMI levels were safety against systolic heart failure in obese individuals than those individuals which have low BMI.

#### DISCUSSION

Different researchers described in their studies that in number of cases obese individuals with greater BMI have healthy response against systolic heart failure. Goel et al. (2011) concluded in their study that concept of obesity paradox is very interesting and blessing for obese people. Clark et al. (2011) were stated in their study that the rate of mortality in greater BMI patients is less than lower BMI patients, because of obesity paradox. In another study the researcher find out that obesity paradox is a biological phenomenon in which obesity provides greater survival and protection in different groups of peoples [18]. However, a lot of studies have since discovered that overweight or obese individuals have a better prognosis than those who are normal weight or underweight [12, 5, and 7].

Same findings are there in this study that cardiac patients with a higher BMI have a better prognosis. Many researchers described in their studies the physiological concept of obesity paradox [13]. Das et al. (2011) concluded that patients having greater BMI have a lot of exercise capacity without any medical complication. In present study patients of Group-A and Group-B regarding BMI showed a significant (P<0.005) changes in oxygen saturation. It was concluded from the above study that further research should focus on determining the impact of obesity on various heart failure syndromes and proper description of obesity paradox in cardiac patients.

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