

HEMODYNAMIC PARAMETERS AND OXYGEN DELIVERY INDEX IN ELDERLY WOMEN WITH CORONARY ARTERY DISEASE AND CHRONIC HEART FAILURE WITH COMORBID OBESITY AND TYPE 2 DIABETES

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ABSTRACT	KEYWORDS
<p>Coronary artery disease (CAD) remains one of the leading causes of morbidity and mortality worldwide, particularly among elderly patients with multiple metabolic comorbidities. Obesity and type 2 diabetes mellitus significantly aggravate the course of CAD and chronic heart failure (CHF) by affecting myocardial metabolism, microcirculation, and oxygen delivery to tissues. Objective: To evaluate the oxygen delivery index (DO₂I) in elderly women with CAD and advanced chronic heart failure (NYHA class III-IV) in the presence of obesity and type 2 diabetes. Methods: A prospective observational cross-sectional study was conducted including 35 women with CAD and CHF class III-IV who were admitted for coronary artery bypass grafting at the Republican Specialized Scientific and Practical Medical Center of Surgery named after Academician V. Vakhidov between January and May 2025. Patients were divided into two groups: CAD+CHF+obesity (n=17) and CAD+CHF+obesity+type 2 diabetes (n=18). A control group consisted of 12 age-matched women without CAD, obesity, or diabetes. Clinical, laboratory, and echocardiographic parameters were assessed. Oxygen delivery index was calculated using the formula $DO_2I = 1.34 \times Hb \times SaO_2 \times CI$. Results: Although hemoglobin levels were comparable between groups, patients with combined obesity and type 2 diabetes demonstrated a significantly lower oxygen delivery index compared with patients with obesity alone (377.5±19.1 vs 437.9±27.7 ml/min/m², p<0.05). Correlation analysis revealed a moderate positive association between DO₂I and hemoglobin levels (r=0.531), heart rate</p>	<p>Coronary artery disease, chronic heart failure, oxygen delivery index, obesity, type 2 diabetes.</p>

($r=0.501$), and LVEDVI ($r=0.435$), while a strong positive correlation was observed with cardiac index ($r=0.870$, $p<0.001$). Conclusion: The coexistence of obesity and type 2 diabetes in elderly women with CHF significantly worsens systemic oxygen delivery. DO_2I may serve as an integrative indicator reflecting combined cardiac dysfunction and metabolic disturbances.

Introduction

Coronary artery disease (CAD) remains one of the leading causes of morbidity and mortality worldwide. According to projections from the American Heart Association (AHA), the prevalence of CAD is expected to increase from 7.8% to approximately 9.2% by 2050. At the same time, the prevalence of metabolic disorders such as type 2 diabetes mellitus and obesity is projected to rise dramatically, from 8% to 26.8% and from 43.1% to 60.6%, respectively [8].

Obesity significantly contributes to the burden of cardiovascular disease. In the United States alone, coronary artery disease associated with obesity was responsible for 273,761 deaths between 1999 and 2022. During this period, age-adjusted mortality rates (AAMR) among individuals with both CAD and obesity increased twofold over 23 years [2]. According to the Global Burden of Disease (GBD) study, approximately 603.7 million adults worldwide are affected by obesity. Moreover, the global number of individuals with obesity doubled between 1973 and 2015, and nearly 39-49% of the world's population (approximately 2.8-3.5 billion people) are currently classified as overweight [12].

Obesity and type 2 diabetes mellitus are not only major cardiometabolic risk factors but also exert direct pathological effects on the myocardium. These metabolic disorders contribute to the development of diabetic cardiomyopathy and coronary microvascular disease (CMVD), which impair myocardial perfusion and oxygen utilization [6,16]. Excess adipose tissue, particularly epicardial fat, can directly affect the coronary microcirculation, which plays a critical role in the regulation of coronary blood flow. Consequently, both obstructive and non-obstructive forms of coronary artery disease are frequently associated with microcirculatory dysfunction [4].

An important integrative parameter reflecting tissue oxygenation is the oxygen delivery index (DO_2I), which represents the combined performance of the respiratory and cardiovascular systems. This indicator characterizes the amount of oxygen delivered to tissues per minute relative to body surface area and therefore provides valuable information about systemic perfusion and oxygen transport capacity [1].

The aim of the present study was to evaluate the oxygen delivery index in patients with coronary artery disease and chronic heart failure (NYHA functional class III-IV) in the presence of obesity and type 2 diabetes mellitus.

Materials and Methods

A prospective observational cross-sectional descriptive study was conducted. The study included 35 female patients diagnosed with coronary artery disease (CAD) and chronic heart failure (CHF) functional class III-IV according to the New York Heart Association (NYHA) classification. All participants were admitted to the Republican Specialized Scientific and Practical Medical Center of Surgery named after Academician V. Vakhidov for coronary artery bypass grafting in the Department

of Surgical Treatment of Coronary Artery Disease and Its Complications between January and May 2025.

Among the examined patients, comorbidity was represented by obesity in 17 women and by obesity combined with type 2 diabetes mellitus in 18 patients. Accordingly, the participants were divided into two clinical groups.

The first group consisted of patients with CAD, CHF, and obesity ($n = 17$). The second group included patients with CAD, CHF, obesity, and type 2 diabetes mellitus ($n = 18$). The control group consisted of 12 women of comparable age without coronary artery disease, obesity, or diabetes mellitus. These individuals were admitted to the Department of Plastic Surgery for elective procedures related to age-associated facial changes.

The study groups were comparable in terms of age and sex distribution. The mean age was 63.0 ± 6.5 years in group 1, 63.1 ± 5.0 years in group 2, and 60.0 ± 6.4 years in the control group ($p > 0.05$). All data are presented as mean values \pm standard deviation.

Obesity was confirmed in all patients of groups 1 and 2, with mean body mass index (BMI) values of 34.6 ± 3.3 kg/m² and 36.8 ± 4.6 kg/m², respectively. Body surface area (BSA) was calculated using the Mosteller formula and averaged 2.0 ± 0.2 m² and 1.9 ± 0.2 m² in groups 1 and 2, respectively ($p > 0.05$). In the control group, BMI was significantly lower (22.1 ± 1.1 kg/m²), with a BSA of 1.69 ± 0.1 m² ($p < 0.05$ compared with both patient groups).

All participants underwent comprehensive clinical and laboratory evaluation including pulse oximetry, complete blood count, lipid profile assessment, and fasting blood glucose measurement.

Echocardiographic examination was performed to assess the following parameters:

- LVEDVI (Left Ventricular End-Diastolic Volume Index), reflecting the degree of left ventricular filling during diastole and normalized to body surface area
- Left ventricular ejection fraction (LVEF)
- Stroke volume (SV)
- Cardiac index (CI)

Cardiac output was calculated as the product of stroke volume and heart rate.

The oxygen delivery index (DO₂I), representing the amount of oxygen delivered to tissues per minute per square meter of body surface area, was calculated using the following formula:

$$\text{DO}_2\text{I} = 1.34 \times \text{Hb} \times \text{SaO}_2 \times \text{CI}$$

This parameter reflects the combined contribution of cardiac output and the oxygen-carrying capacity of blood.

Statistical analysis was performed using **MedCalc software version 23.2.1**.

Results

The analysis showed that hemoglobin concentration did not differ significantly between the two patient groups and the control group. However, hemoglobin levels in group 2 (patients with CAD, CHF, obesity, and type 2 diabetes) were slightly lower than in group 1 (121.2 ± 11.6 g/L vs. 125.2 ± 18.1 g/L), although this difference did not reach statistical significance ($p > 0.05$). Considering that each gram of hemoglobin is capable of transporting approximately 1.34 mL of oxygen, even minor differences in hemoglobin levels may contribute to variations in systemic oxygen delivery.

Due to the presence of chronic heart failure, echocardiographic parameters reflecting cardiac structure and function differed significantly between the patient groups and the control group. Nevertheless, no statistically significant differences were observed between group 1 and group 2 with regard to left

ventricular ejection fraction (LVEF), stroke volume, cardiac index, or heart rate. It should be noted that patients in group 2 demonstrated slightly higher mean heart rate values, whereas LVEF, stroke volume, and cardiac index tended to be somewhat lower compared with group 1, although these differences were not statistically significant ($p > 0.05$).

Despite the absence of significant differences in individual hemodynamic parameters between the two patient groups, the combined effect of reduced cardiac functional capacity and lower hemoglobin levels in patients with CAD, CHF, obesity, and type 2 diabetes had a substantial impact on the oxygen delivery index (DO₂I). As a result, DO₂I was significantly lower in group 2 compared with group 1 ($p < 0.05$) (Table 1, Figure 1).

Table 1. Instrumental and Laboratory Parameters of the Studied Groups

Parameter	Control	Group 1	Group 2	p (Control vs Group 2)	p (Group 1 vs Group 2)
LVEF (%)	73.7 ± 1.3	54.5 ± 8.5	51.8 ± 9.6	<0.05	>0.05
Stroke Volume (mL)	84 ± 4.8	71.1 ± 23.5	66.9 ± 16.1	<0.05	>0.05
Heart Rate (beats/min)	68.3 ± 3.5	74.7 ± 16.6	80.2 ± 12.9	>0.05	>0.05
Body Surface Area (m ²)	1.69 ± 0.02	1.9 ± 0.2	2.0 ± 0.2	<0.05	>0.05
Cardiac Index (L/min/m ²)	3.39 ± 0.32	2.76 ± 0.74	2.68 ± 0.44	<0.05	>0.05
SaO ₂	0.99 ± 0.12	0.95 ± 0.01	0.95 ± 0.01	<0.05	>0.05
Hemoglobin (g/L)	125.8 ± 11.3	125.2 ± 18.1	121.2 ± 11.6	>0.05	>0.05
DO ₂ I (mL/min/m ²)	566.5 ± 9.1	437.9 ± 27.7	377.5 ± 19.1	<0.05	<0.05

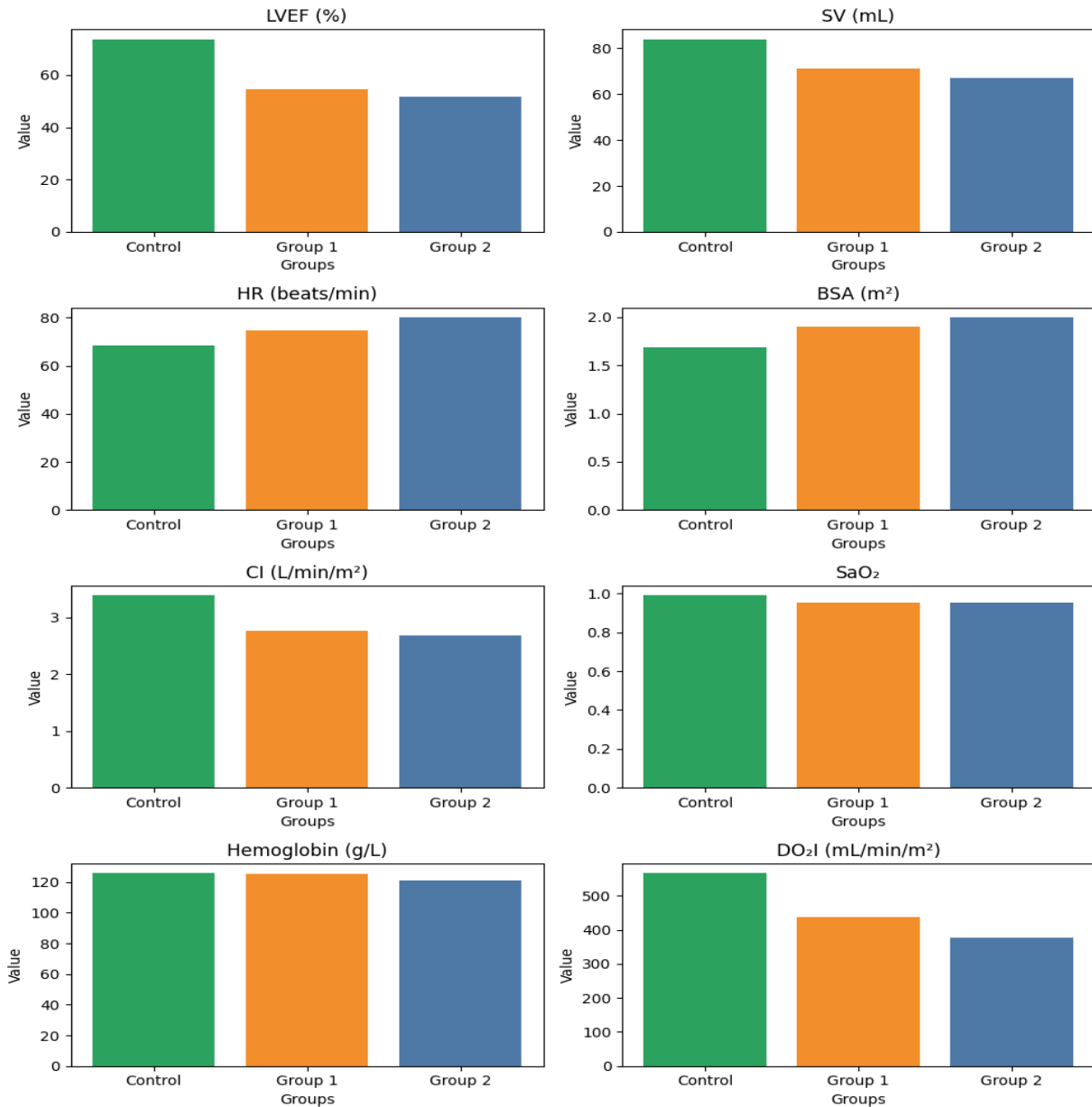


Figure 2*. Comparative analysis of hemodynamic and laboratory parameters among the study groups.

Green bars represent the control group, orange bars represent patients with coronary artery disease, chronic heart failure, and obesity (Group 1), and blue bars represent patients with coronary artery disease, chronic heart failure, obesity, and type 2 diabetes mellitus (Group 2).

*The figure illustrates differences in left ventricular ejection fraction (LVEF), stroke volume (SV), heart rate (HR), body surface area (BSA), cardiac index (CI), oxygen saturation (SaO₂), hemoglobin level (Hb), and oxygen delivery index (DO₂I) between the control group and patients with coronary artery disease and chronic heart failure with obesity and type 2 diabetes mellitus.

The obtained results indicate that the oxygen delivery index in both patient groups was located at the lower limit of the reference range (360-500 mL/min/m²). Critical reduction of this parameter below 300 mL/min/m² is known to be associated with increased mortality. These findings suggest impairment not only in the quantity of oxygen delivered to tissues due to reduced cardiac index but also in the

quality of oxygen transport resulting from decreased oxygen-carrying capacity of blood and disturbances in microcirculation and tissue oxygen utilization.

Patients in group 2 demonstrated greater variability in several parameters, which may reflect the presence of endothelial dysfunction and metabolic disturbances associated with diabetes mellitus. Increased LVEDVI and reduced LVEF indicate volume overload and dilation of the left ventricle, which are commonly observed in patients with obesity and diabetes.

In addition, oxygen saturation in patients of groups 1 and 2 decreased to approximately 95%. This finding may be explained by several factors, including pulmonary congestion associated with chronic heart failure, hypoventilation-particularly in the supine position-and microvascular abnormalities related to diabetes mellitus, which may impair alveolar perfusion and tissue oxygenation. Although hemoglobin and SpO₂ values remained within the lower limit of the reference range, their combined effects contributed to the observed decrease in DO₂I.

Thus, in both patient groups the left ventricle was unable to maintain sufficient cardiac output to ensure adequate systemic oxygen delivery. The increase in heart rate observed in group 2 did not compensate for reduced cardiac performance and oxygen transport capacity. Consequently, these alterations were reflected in a significant reduction of DO₂I in patients with CAD, CHF, obesity, and type 2 diabetes compared with those with obesity alone ($p < 0.05$).

The oxygen delivery index reflects the combined impact of impaired cardiac function as well as metabolic disturbances associated with obesity and diabetes mellitus on tissue oxygenation. In patients with CHF, obesity, and type 2 diabetes, DO₂I was significantly lower than in patients with CHF and obesity alone. This finding may be explained by several mechanisms, including diabetic cardiomyopathy, anemia of chronic disease, reduced erythropoiesis related to diabetic nephropathy, and diabetic microangiopathy.

Correlation analysis demonstrated a moderate positive relationship between DO₂I and hemoglobin level ($r = 0.531$), heart rate ($r = 0.501$), and LVEDVI ($r = 0.435$). A strong positive correlation was observed between DO₂I and cardiac index ($r = 0.870$) (Table 2).

Table 2. Correlation Between DO₂I and Parameters Influencing Tissue Oxygenation

Parameter	Correlation coefficient (r)	p-value
LVEF	-0.098	0.6409
Hemoglobin	0.531	0.0063
Heart Rate	0.501	0.0107
LVEDVI	0.435	0.03
SpO ₂	0.313	0.1275
Body Weight	-0.076	0.7188
Age	-0.172	0.4115
Body Surface Area	-0.11	0.6022
Height	-0.28	0.1753
Cardiac Index	0.870	<0.0001

Correlation analysis revealed a strong positive relationship between the oxygen delivery index (DO₂I) and the cardiac index (CI) ($r = 0.870$, $p < 0.0001$). This relationship is illustrated in Figure 2.

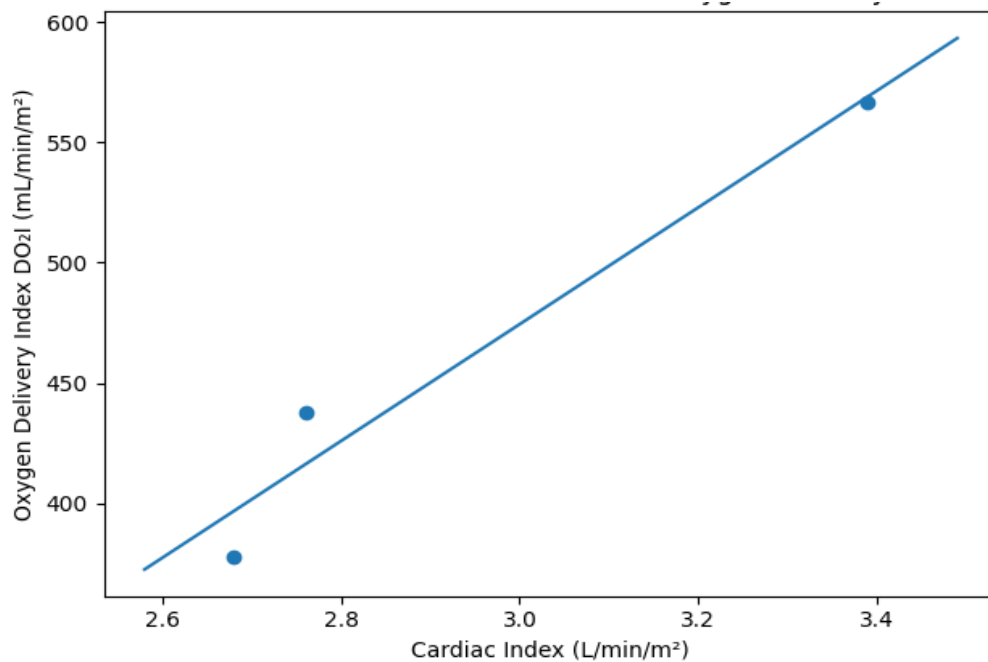


Figure 3. Correlation between cardiac index and oxygen delivery index (DO₂I).

A strong positive association was observed between cardiac index and oxygen delivery index, indicating that reduced cardiac output significantly contributes to impaired systemic oxygen delivery in patients with coronary artery disease and chronic heart failure.

Discussion

The present study demonstrates the significant contribution of obesity and type 2 diabetes mellitus to the reduction of the oxygen delivery index (DO₂I) in patients with chronic heart failure (CHF) of NYHA functional class III-IV. The obtained findings are consistent with contemporary literature describing the negative impact of metabolic disorders on cardiovascular outcomes. In particular, it has been reported that an increased triglyceride-glucose index is associated with a higher risk of mortality in patients with coronary artery disease and metabolic comorbidities (HR = 1.21; 95% CI 1.02-1.34; $p < 0.01$) [22].

In patients with obesity and type 2 diabetes, chronic heart failure is frequently accompanied by coronary microvascular disease (CMVD), which is closely related to endothelial dysfunction and structural remodeling of small coronary vessels [19]. These microvascular alterations impair myocardial perfusion and contribute to the progression of cardiac dysfunction.

Recent molecular studies have also provided insights into the mechanisms linking obesity-related metabolic disturbances with coronary artery disease. Proteomic analyses and RNA sequencing have identified endotrophin, a C-terminal peptide fragment of collagen VI alpha-3 (COL6A3), as an important biomarker associated with myocardial injury. Endotrophin levels increase proportionally with body mass index and decrease following weight reduction, reflecting the degree of myocardial involvement in obesity-related cardiovascular pathology [20].

Another potential molecular link between metabolic disorders and coronary artery disease involves mitochondrial dysfunction. Reduced mitochondrial DNA levels have been observed in patients with

dyslipidemia and coronary artery disease, suggesting that mitochondrial DNA may serve as a biomarker reflecting the relationship between metabolic disturbances and myocardial damage [13].

In addition, increasing attention has been given to irisin, a biologically active myokine involved in energy metabolism. Irisin is expressed in multiple tissues and is stimulated by physical activity and exposure to cold. It plays a role in mitochondrial respiratory regulation, reduces the generation of reactive oxygen species, and modulates inflammatory responses. Reduced irisin levels have been reported in individuals with metabolic syndrome, aging, and obesity, as well as in patients with coronary artery disease and stroke [23].

Population studies indicate that Asian populations may have a higher susceptibility to type 2 diabetes mellitus due to genetic predisposition combined with environmental and lifestyle factors [21]. These include genetic variations affecting enzymes of energy metabolism, lipid metabolism, antioxidant defense mechanisms, DNA repair systems, and endothelial function [11]. The interaction of these genetic factors with environmental influences such as smoking, sedentary lifestyle, and dietary patterns contributes cumulatively to the development of cardiometabolic disease [21].

Objective assessment of myocardial condition can also be obtained using biomarkers such as NT-proBNP. Data from the ARISE-HF study demonstrated that patients with diabetic cardiomyopathy exhibited significantly higher levels of NT-proBNP and high-sensitivity troponin compared with individuals without diabetes mellitus [7]. These findings support the role of NT-proBNP as an early marker of subclinical diabetic cardiomyopathy [10,14].

Furthermore, studies have shown that even in the preclinical stage of cardiac dysfunction, NT-proBNP levels may not correlate directly with echocardiographic parameters but instead reflect reduced physical activity and functional capacity. Significant correlations have been observed with clinical scales such as the Kansas City Cardiomyopathy Questionnaire score, the Physical Activity Scale in the Elderly, exercise duration during cardiopulmonary testing, peak oxygen consumption (VO_2), and ventilatory efficiency indices [6].

Another important factor contributing to reduced oxygen delivery in our cohort is aging. Most patients in the present study were older than 60 years. Aging is associated with several metabolic changes, including a shift in energy metabolism from carbohydrate oxidation toward lipid utilization, development of insulin resistance, mitochondrial dysfunction, and accumulation of lipids within cardiomyocytes [5].

Age-related cardiac alterations include myocardial hypertrophy, interstitial fibrosis, and impaired contractile function [18]. In addition, chronic low-grade systemic inflammation and altered inflammatory mediator profiles further aggravate myocardial dysfunction by promoting immune cell infiltration within the myocardium and contributing to progressive cardiac remodeling [15,17].

Taken together, these mechanisms explain the observed reduction in DO_2I among patients with chronic heart failure complicated by obesity and type 2 diabetes mellitus. The combination of metabolic disturbances, endothelial dysfunction, and structural myocardial changes results in impaired systemic perfusion and reduced tissue oxygenation.

Importantly, symptoms such as dyspnea, fatigue, and peripheral edema may often be attributed by patients to obesity itself, which may delay the recognition of worsening heart failure. Consequently, the clinical course of CHF in obese patients with diabetes may appear less typical yet be associated with a more unfavorable prognosis. This highlights the importance of careful clinical assessment, the use of instrumental diagnostic methods, and active management of metabolic disorders.

Early detection of hemodynamic signs of impaired systemic perfusion related to reduced oxygen delivery is therefore essential for optimizing therapeutic strategies in patients with chronic heart failure complicated by obesity and type 2 diabetes mellitus. The present data suggest that even modest reductions in cardiac index, slight decreases in hemoglobin concentration affecting blood oxygen capacity, and alterations in LVEDVI may serve as potential predictors of reduced DO₂I.

Study Limitations

Despite the valuable findings obtained in this study, several limitations should be acknowledged. First, the study was conducted on a relatively small sample size and included only female patients, which may limit the generalizability of the results to the broader population. Second, the study had a cross-sectional design, which restricts the ability to establish causal relationships between metabolic disorders and changes in oxygen delivery index. Third, although comprehensive clinical and laboratory assessments were performed, additional biomarkers associated with metabolic and inflammatory pathways were not evaluated. Future studies involving larger cohorts, including both male and female patients, as well as longitudinal follow-up, are required to further clarify the mechanisms linking metabolic disorders, chronic heart failure, and systemic oxygen delivery.

Clinical Implications

The results of the present study highlight the importance of assessing systemic oxygen delivery in patients with chronic heart failure complicated by obesity and type 2 diabetes mellitus. The oxygen delivery index represents an integrative physiological parameter reflecting the combined effects of cardiac function, blood oxygen-carrying capacity, and systemic perfusion. Early identification of reduced DO₂I may help clinicians detect subclinical deterioration of hemodynamic status and optimize therapeutic strategies. Comprehensive management of metabolic disorders, including weight reduction, glycemic control, and correction of anemia, may contribute to improved tissue oxygenation and potentially better clinical outcomes in this patient population.

Conclusions

1. Structural and functional cardiac parameters, hemoglobin concentration, and oxygen saturation did not demonstrate statistically significant differences between women with chronic heart failure (NYHA class III-IV) and obesity and those with chronic heart failure combined with obesity and type 2 diabetes mellitus.
2. The oxygen delivery index was significantly lower in patients with chronic heart failure associated with obesity and type 2 diabetes mellitus compared with patients with obesity alone (377.5 ± 19.1 mL/min/m² vs. 437.9 ± 27.7 mL/min/m², $p < 0.05$).
3. Correlation analysis revealed moderate positive relationships between DO₂I and hemoglobin level ($r = 0.531$, $p < 0.05$), heart rate ($r = 0.501$, $p < 0.05$), and LVEDVI ($r = 0.435$, $p < 0.05$), as well as a strong positive correlation between DO₂I and cardiac index ($r = 0.870$, $p < 0.05$).
4. The oxygen delivery index represents an integrative physiological parameter reflecting the combined influence of structural and functional cardiovascular abnormalities in chronic heart failure together with metabolic disturbances associated with obesity and type 2 diabetes mellitus, ultimately leading to impaired systemic perfusion and reduced tissue oxygenation.

References

1. Naumov AB, Makaruk VV, Marchenko SP, Chupaeva OYu, Volkov AM, Khubulava GG. Calculation of left ventricular stroke volume using the Relo formula: correct geometric characteristics of the aortic root in the assessment of systemic blood flow. *Vestnik Khirurgii imeni I. I. Grekova*. 2022;181(3):12-19. doi:10.24884/0042-4625-2022-181-3-12-19. (In Russian)
2. Ahmed Mushood, Hira Javaid, Aimen Shafiq Trends and Disparities in Coronary Artery Disease and Obesity- Related Mortality in the United States From 1999-2022 // *Endocrinology, Diabetes & Metabolism*,2024; 7:e70010 <https://doi.org/10.1002/edm2.70010>.
3. Duggan John P , Alex S Peters, Gregory D Trachiotis, Jared L Antevil Epidemiology of Coronary Artery Disease // *Surg Clin North Am* - 2022 Jun;102(3):499-516. doi: 10.1016/j. suc.2022.01.007.
4. Favalaro Liliana E , Roxana D Ratto, Carla Musso Heart Failure and Diabetes: Perspective of a Dangerous Association // *Curr Hypertens Rev* . 2021;17(2):85-93.doi: 10.2174/1573402117666210406111927
5. Fang Zhuyubing, Umar Raza, Jia Song Systemic aging fuels heart failure: Molecular mechanisms and therapeutic avenues // *ESC Heart Failure*2025;12: 1059-1080 Published online 22 July 2024 in Wiley Online Library (wileyonlinelibrary.com)DOI:10.1002/ehf2.14947/
6. Gouda Pishoy, Yuxi Liu , Javed Butler Relationship between NT-proBNP, echocardiographic abnormalities and functional status in patients with subclinical diabetic cardiomyopathy // *Cardiovasc Diabetol* - 2024 Aug 2;23(1):281.doi: 10.1186/s12933-024-02378-w
7. Januzzi James L , Stefano Del Prato, Julio Rosenstock Characterizing diabetic cardiomyopathy: baseline results from the ARISE-HF trial // *Cardiovasc Diabetol* - 2024 Feb 1;23(1):49. doi: 10.1186/s12933-024-02135-z.
8. Maddox Karen E Joynt , Mitchell S V Elkind, Hugo J Aparicio, Yvonne Commodore-Mensah, Sarah D de Ferranti, William N Dowd, Adrian F Hernandez, Olga Khavjou, Erin D Michos, Latha Palaniappan, Joanne Penko, Remy Poudel, Véronique L Roger, Dhruv S Kazi; American Heart Association Forecasting the Burden of Cardiovascular Disease and Stroke in the United States Through 2050-Prevalence of Risk Factors and Disease: A Presidential Advisory From the American Heart Association // *Circulation*. -2024 Jul 23;150(4):e65-e88. doi: 10.1161/CIR.0000000000001256. Epub 2024 Jun 4.
9. Marzà-Florensa Anna , Ilonca Vaartjes, Ian Graham, Kerstin Klipstein-Grobusch, Diederick E Grobbee; Surf CHD II collaborators. A Global Perspective on Cardiovascular Risk Factors by Educational Level in CHD Patients: SURF CHD II // *Glob Heart* - 2024 Jul 16;19(1):60. doi: 10.5334/gh.1340. eCollection 2024.
10. McEvoy John William , Catriona Jennings, Kornelia Kotseva Variation in secondary prevention of coronary heart disease: the INTERASPIRE study Multicenter Study // *Eur Heart J*.- 2024 Oct 14;45(39):4184-4196. doi: 10.1093/eurheartj/ehae558.
11. Miao Ying Yu Wang, <https://pubmed.ncbi.nlm.nih.gov/40087495/> - full-view-affiliation-7 Qin Wan Association between TyG index with obesity indicators and coronary heart disease: a cohort study // *Sci Rep* - 2025 Mar 14;15(1):8920. doi: 10.1038/s 41598-025-93922-5.
12. Powell-Wiley Tiffany M , Paul Poirier, Lora E Burke Obesity and Cardiovascular Disease: A Scientific Statement From the American Heart Association // *Circulation* - 2021 May 25;143(21):e984-e1010.

13. Qin Pei , Tianhang Qin , Lei Liang , Xinying Li , Bin Jiang , Xiaojie Wang, Jianping Ma, Fulan Hu Ming Zhang Dongsheng Hu The role of mitochondrial DNA copy number in cardiometabolic disease: a bidirectional two-sample mendelian randomization study // *Cardiovasc Diabetol* - 2024 Jan 28;23(1):45. doi: 10.1186/s12933-023-02074-1.
14. Report on Cardiovascular Health and Diseases in China 2021: An Updated Summary // *Biomed Environ Sci* - 2022 Jul 20;35(7):573-603. doi: 10.3967/bes2022.079.
15. Seferović Petar M , Walter J Paulus, Giuseppe Rosano Diabetic myocardial disorder. A clinical consensus statement of the Heart Failure Association of the ESC and the ESC Working Group on Myocardial & Pericardial Diseases // *Eur J Heart Fail* - 2024 Sep;26(9):1893-1903. doi: 10.1002/ejhf.3347. Epub 2024 Jun 19
16. Stanton Ana Maria , Muthiah Vaduganathan, Lee-Shing Chang , Alexander Turchin, James L Januzzi Jr, Vanita R Aroda. Asymptomatic Diabetic Cardiomyopathy: an Underrecognized Entity in Type 2 Diabetes // *Curr Diab Rep* - 2021 Sep 27;21(10):41. doi: 10.1007/s11892-021-01407-2.
17. Syed Akheel A , Safwaan Adam, Christopher A Miller, Bilal Alkhaffaf Obesity Management for Patients with Coronary Artery Disease and Heart Failure // *Heart Fail Clin* -2025 Apr;21(2):257-271. doi: 10.1016/j.hfc.2024.12.006. Epub 2025 Feb 7.
18. Triposkiadis Filippos , Andrew Xanthopoulos, John Parissis, Javed Butler, Dimitrios Farmakis Pathogenesis of chronic heart failure: cardiovascular aging, risk factors, comorbidities, and disease modifiers // *Heart Fail Rev* - 2022 Jan;27(1):337-344. doi: 10.1007/s10741-020-09987-z.
19. Xiaoling Cai, Wenjia Yang Bo Feng Qiuhe Ji, Ming Liu, Yanbing Li, Nanwei Tong, Ningling Sun, Minghui Zhao, Linong Ji. Real-World Multicenter Registry to Determine Management and Quality of Care of Patients with Type 2 Diabetes, Hypertension, Heart Failure and/or Chronic Kidney Diseases in China (iCaReMe China) // *Adv Ther* - 2025 Mar;42(3):1600-1609. doi: 10.1007/s12325-025-03114-w. Epub 2025 Feb 10
20. Yoshiji Satoshi Tianyuan Lu , Guillaume Butler-Laporte Integrative proteogenomic analysis identifies COL6A3-derived endotrophin as a mediator of the effect of obesity on coronary artery disease // *Nat Genet.* - 2025 Feb;57(2):345-357. doi: 10.1038/s41588-024-02052-7. Epub 2025 Jan 24
21. Zarkasi Khairul Anwar, Nor Azian Abdul Murad, Norfazilah Ahmad, Rahman Jamal, Noraidatulakma Abdullah Coronary Heart Disease in Type 2 Diabetes Mellitus: Genetic Factors and Their Mechanisms, Gene-Gene, and Gene-Environment Interactions in the Asian Populations // *Int J Environ Res Public Health*- 2022 Jan 6;19(2):647. doi: 0.3390/ijerph19020647
22. Zeng Xiao , Yuping Liu, Ping Shuai, Peiyuan He, Xiaoli Liu The relationship and differences in the triglyceride-glucose index and all-cause mortality in patients with coronary heart disease combined with cerebrovascular and other comorbidities: an analysis of the MIMIC-IV database // *Front Cardiovasc Med.*-2025 Apr 9;12:1572709. doi: 10.3389/fcvm.2025.1572709. eCollection 2025.
23. Zhang Hu , Xiaowen Wu, Jiling Liang, Michael Kirberger, Ning Chen Irisin, an exercise-induced bioactive peptide beneficial for health promotion during aging process // *Ageing Res Rev* - 2022 Sep;80:101680. doi: 10.1016/j.arr.2022.101680. Epub 2022 Jul 3.