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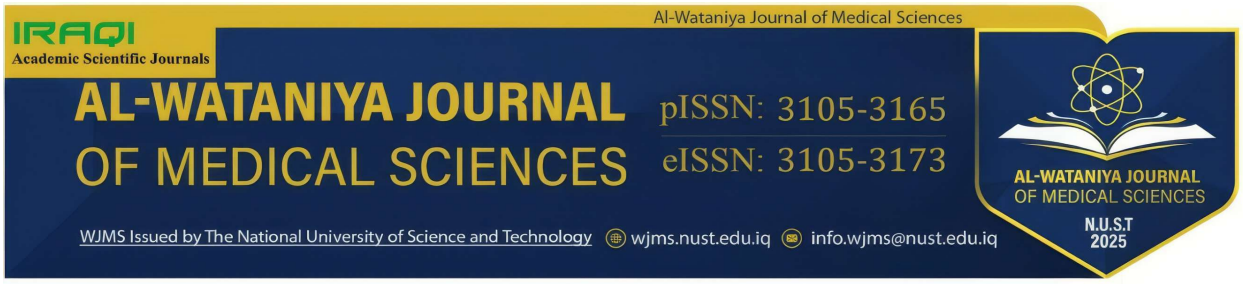

Article

Assessment of Serum HSP20 Levels and Lipid Profile Alterations in Patients with Obesity and Cardiovascular Disease

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Abstract

Background: As a major risk factor for a number of cardiovascular illnesses (CVDs), such as hypertension and heart failure, obesity is a major global health concern. The purpose of this study was to examine the metabolic profiles and function of Heat Shock Protein 20 (HSP20) in patients with CVD and obesity.

Methods: 43 people (aged 13 to 70) participated in a clinical trial at Nasiriya General Hospital. They were divided into three group: CVD patients (n=18), obese people (n=15), and healthy controls (n=10). Important bio marks were examined, such as blood glucose, lipid profile and HSP20.

Results: The results showed that the obese group had a substantially higher BMI (31.17\0.92). LDL levels increased by 15.3% and HDL levels decreased by 12.7% in patients with CVD. On the other hand, those who were obese had triglyceride levels that were 18.5% higher. The prevalence of smoking among CVD patients was 57.7%, and both groups had elevated blood pressure and blood glucose. Notably, both obese and CVD participants had noticeably higher HSP20 levels.

Conclusions: This study shows that a key risk factor for CVD in the participants is obesity induced metabolic dysregulation, which is marked by notable changes in lipid profiles and high blood glucose. The results show that these risks are much increased when smoking and hypertension coexist, which accelerates vascular problems. HSP20 is a crucial biomarker of physiological stress and a putative cardio protective mediator, as evidenced by the notable rise of serum HSP20 in both obese and CVD patients. Therefore, the best approach for reducing cardiovascular morbidity and using HSP20 for early diagnostic evaluation is still to treat obesity through integrated clinical and lifestyle therapies.

Keywords: Obesity, CVDs, HSP20, Body Mass Index, Lipid Profile

1. Introduction

Excessive fat accumulation that poses serious dangers to general health is the hallmark of obesity, a major global health concern. A body mass index (BMI) of 30 or more is considered obese by the World Health Organization (WHO). The high correlation between obesity and cardiovascular illness (CVDs), such as heart failure, hypertension, and coronary artery disease, is one of the most dangerous effects of obesity [1].

Overweight, especially visceral fat, increases the risk of heart disease by fostering metabolic conditions such as insulin resistance, dyslipidemia, and chronic inflammation. Atherosclerosis, a disorder where fatty deposits accumulate in the arteries, narrows them, and raises the risk of heart attacks and strokes, is brought on by several

variables. Furthermore, obesity raises blood pressure and puts more strain on the heart, both of which can result in long-term cardiac problems [2].

Furthermore, the condition of persistent low-grade inflammation is part of the complex pathophysiology of obesity-induced cardiovascular problems. Previously thought of as just a storage facility, adipose tissue is now understood to be an active endocrine organ that secretes a variety of pro-inflammatory cytokines. The deregulation of these variables in obese people leads to endothelial dysfunction and faster plaque development in the artery walls. Together with oxidative stress, this inflammatory milieu severely reduces vascular responsiveness and accelerates the development of atherosclerosis, raising the clinical risk of myocardial infraction and stroke [3].

A member of the small heat shock protein family, Heat Shock Protein 20 (HSP20), sometimes referred to as HSPB6, serves as a molecular chaperone to preserve cellular homeostasis in stressful situations. HSP 20 is essential for the cardiovascular system because it protects cardio myocytes, encourages vasodilation, and prevents platelet aggregation. According to recent research, metabolic dysregulation, such as obesity and insulin resistance, may cause a considerable modulation of HSP20 levels. HSP20 has become a promising circulating biomarker for the early identification and tracking of cardiovascular problems due to its role in inflammatory pathways and lipid metabolism. To comprehend its diagnostic and prognostic usefulness in these related illnesses, it is crucial to examine its levels in individuals with obesity and CVD.

A balanced diet frequent exercise is important lifestyle changes for controlling obesity-related heart disease, but advanced therapeutic management requires an understanding of molecular biomarkers. With increased levels seen in both CVD and obese people, Heat Shock Protein 20 (HSP20) has become a prominent biomarker that may play a part in both disease development and prevention [4].

2. Materials and Methods

2.1 Study Design

Nasiriya General Hospital served as the site of this clinical investigation. There were 43 participants in the study, which used a cross-sectional design. The participants were divided into three main groups: those with cardiovascular disease (CVD) (n=18), those who were obese (n=15), and a healthy control group (n=10). The participants ages varied from 13 to 70.

Patients with a formal diagnosis of obesity and cardiovascular disease were included in the study. To replicate real-world clinical presentations, participants with related variables such as smoking, a history of stroke, acute episodes, or prior COVID-19 infection were also included.

Exclusion criteria, people with significant comorbidities, such as cancer or chronic kidney disease, were not included in the study to preserve data integrity. Patients with severe mental illnesses, recent surgical trauma, acute infections (other than COVID-19), or metabolic diseases unrelated to obesity were also excluded. Additionally, pregnant women were excluded from the sample.

2.2 Materials

Sterilized syringes were used to draw five ml of venous blood samples. After being put into gel tubes with labels, the blood was left to coagulate for ten minutes at room temperature. The samples were centrifuged for 15 minutes at 6000rpm to separate the serum. After that, the isolated serum was divided into Eppendorf tubes and kept at -80 °C to maintain the stability of the biomarker for further laboratory examination.

As explained below, premium diagnostic kits and specialist equipment were used for all biochemical analyses:

- Lipid profile and Glucose: Biorex(Germany) enzymatic kits were used to measure the lipid components of serum and Random Blood Sugar (RBS).
- HSP20 Quantification: An Enzyme-Linked Immunosorbent Assay (ELISA) kit from SUNLONG (China) (Cat-NO. E-EL H0066) was used to measure the amounts of Human Heat Shock Protein 20(HSP20).
- Clinical Equipment: An Omron BP742N sphygmomanometer (USA) was used to measure blood pressure.
- A Siemens hematological analyzer (Germany) were additional necessary instruments.

2.3 Analytical Methods

All The following is how the clinical and biochemical parameters were determined and examined:

- Human anthropometry measurements include: the following formula was used to determine the body mass index;

$$BMI = \frac{\text{Weight (kg)}}{\text{Height (m)}^2} \text{ (Eq1)}$$

The World Health Organizations physical status standards used as the basis for classification.

- Clinical Assessment: the average of two independent measures performed at 5-minutes intervals was used to record blood pressure following a 5- minutes seated rest period
- Biochemical analysis: enzymatic tests for HDL-C, triglycerides, and cholesterol were used to evaluate lipid profiles. Using the glucose oxidase technique, which measures changes in absorbance after enzymatic reactions, blood glucose was determined.
- Statistical processing: SPSS software (version 26, IBM, USA) was used to analyze the data. To ascertain statistical difference between the study groups, a One-Way Analysis of Variance (ANOVA) was utilized. When the P-value was less than 0.05, all results were deemed statistically significant.

2.4 Ethical Approval

All participants provided informed consent prior to their inclusion in the study. Personal data and clinical results were handled with strict confidentiality.

3. Results and Discussion

3.1 Demographic characteristics

A total of 43 cases-18 CVD patients, 15 obese people, and 10 controls- were analyzed in this study. The patients ages varied, as indicated in Table (1), with CVD patients having the greatest mean age (55.81± 3.257 years), followed by the control group (34.11±5.478years) and obese patients (37.67±3.125 years). Obese patients had a significantly higher BMI (35.70±0.775kg/m2). While 46.2% of CVD patients were overweight, the majority of obese people (84.6%) were classified as obese.

Both the CVD and control groups had equal percentages of males and females (50% each), while the obese group had a greater number of males (58% male, 42% female). The CVD group had the highest smoking prevalence (58%), followed by obese people (33%), whereas none of the control subjects smoked. According to a prior study, smoking is the most common preventable cause of cardiovascular disease (CVD), which leads to heart failure and atherosclerosis [5].

Cardiovascular problems account for two-thirds of obesity-related increased mortality, indicating a causal relationship between obesity and CVD. Studies have shown that the combination of smoking and obesity can exacerbate cardiovascular health by increasing cardiovascular dysfunction [6].

Table 1: Comparison of the clinical characteristics between CVD Patients, obesity, and control groups

Clinical characteristics	Mean ± SE		
	CVD Patient N=18	OBESITY N=15	Control N=10
Age (year)	55.81± 3.257	37.67 ± 3.125	21.6 ± 1.2
	A	B	c
BMI (kg/m2)	25.86 ± 0.775	35.70 ± 0.878	21.57±1.03
Normal weight	(38.5%)	(0%)	(100%)
Overweight	(46.2%)	(15.4 %)	c
obese	(15.4%)	(84.6%)	
	A	B	
Gender	Male (50 %)	Male (58 %)	Male (50 %)
	Female (50 %)	Female (42 %)	Female (50 %)

Smoking	Yes (58 %) No (42 %)	Yes (33 %) No (67 %)	No (100%)

*** P< 0.05 statistically significant with the control group**

This study found that both CVD and control groups had an equal gender distribution (50% male and female), while the obesity group had a higher male proportion (58%). Obesity rates in older men increased from 31.6% to 41.5% between 1999 and 2010, with projections suggesting that 58% of females and 57% of males born between 1984 and 1988 will be obese by age 55 [7].

Over the past 40 years, obesity rates among older persons have tripled, particularly in both industrialized and developing nations. Sarcopenia and other age-related physiological changes lead to sarcopenic obesity. Which complicates health consequences. Increased morbidity and death, especially from cardiovascular [8].

3.2 Lipid profile

Table 2 displays the mean ± SE of the comparison of the lipid profiles of CVD patients, obese, and control group. The levels of obesity (231.42±14.339) and CVD patients (195.04±7.594) were substantially higher than those of the control group (148.44±7.073) (P>0.05). Triglyceride levels were substantially higher in CVD patients (257.31±16.377) and obese individuals (390.67±28.545) than in the control group 9146.11±5.978) (P>0.05). HDL values were significantly lower in CVD patients (47.56±3.520) and obese individuals (50.81±3.130) than in the control group (58.00±4.708) (P>0.05). LDL values were considerably higher in CVD patients (143.54±8.763) and obese individuals (161.17±14.943) than in the control group (90.67±3.555) (P>0.05). When compared to controls, these results show notable anomalies in the lipid profiles of the obese and CVD groups.

Table 2: Comparison of the clinical characteristics between CVD Patients, obesity, and control groups

Lipid profile	**Mean ± SE**		
	CVD Patient N=18	**OBESITY N=15**	**Control N=10**
Cholesterol (mg/dl)	195.04 ± 7.594 A	231.42± 14.339 b	148.44 ± 7.073 C
Triglyceride (mg/dl)	257.31 ±16.377 A	390.67 ± 28.545 b	146.11±5.978 C
HDL (mg/dl)	47.56±3.520	50.81±3.130	58.00±4.708
LDL (mg/dl)	143.54±8.763 A	161.17±14.943 a	90.67±3.555 B

*** P< 0.05 statistically significant with the control group**

In comparison to the control group (148.44±7.073), the current investigation found considerably higher levels of important cardiovascular risk factors in both CVD patients (195.04±7.594) and those with obesity (231.42±14.339), with a statistically significant difference(P>0.05). Furthermore, triglyceride levels were substantially higher in CVD patients (257.31±16.377) and obese adults (390.67±28.545) than in the control group (146.11± 5.978) (P>.0.05). These results are in line with other studies showing that dyslipidemia- specifically, high levels of cholesterol and triglycerides-is essential to the onset and advancement of cardiovascular disease (CVD).

One known risk factor for atherosclerotic cardiovascular disease (ASCVD) is elevated cholesterol, namely high LDL-C and low HDL-C. elevated LDL-C levels promote the development of plaque in the artery walls, which narrows the arteries and raises the risk of heart attacks and strokes [9]. Every year, high blood cholesterol cause around 4.4 million deaths, with ischemic strokes and coronary heart disease (CHD) being the main causes of these deaths [10].

While dietary cholesterols effect on blood cholesterol levels used to be the main focus of research, new findings indicate that saturated fats have more important role in rising cholesterol levels and increasing the risk of CVD. Dietary guidelines and the general treatment of cardiovascular health are affected by this change in knowledge [11].

Furthermore, through processes like the release of free fatty acids, the generation of proinflammatory cytokines, and the impairment of fibrinolysis, hypertriglyceridemia-another important characteristic seen in both the obese and CVD groups- can exacerbate CVD [12]. Furthermore, the triglyceride-glucose index has become a unique predictor of cardiovascular events, indicating that the risk of CVD is further increased by insulin resistance linked to excessive triglyceride levels [13]. These results highlight the significance of early intervention and lipid profile management, especially in obese person to reduce the risk of cardiovascular morbidity and mortality.

In this study, we found that CVD patients (47.56±3.520) and obese people (50.81±3.130) had significantly lower levels of HDL cholesterol than the control group (58.00±4.708) ($P>0.05$). Furthermore, the obese and CVD groups showed noticeably greater. Low density lipoprotein (LDL) cholesterol levels were 143.54±8.763 for CVD patients and 161.17±14.943 for obese patients in comparison to the control group (90.67±3.555) ($P>0.05$). These results underscore the importance of dyslipidemia in the pathophysiology of cardiovascular illness by highlighting notable anomalies in lipid profiles in both the obese and CVD groups.

Low density lipoprotein (LDL) cholesterol levels were 143.54±8.763 for CVD patients and 161.17±14.943 for obese patients in comparison to the control group (90.67±3.555) ($P>0.05$). These results underscore the importance of dyslipidemia in the pathophysiology of cardiovascular illness by highlighting notable anomalies in lipid profiles in both the obese and CVD groups.

Due to several biological and environmental variables, an increase in LDL-C is a significant factor in the development of cardiovascular disease (CVD). It has been demonstrated that tumor necrosis factor-alpha (TNF-α) causes the release of soluble LDL receptors (s LDL-R), which prevent LDL-C from being cleared by the liver and raise the levels of LDL-C in the blood. Furthermore, by raising LDL-R levels, chronic inflammation-which is prevent in obesity and CVD- can worsen LDL buildup in the blood. The development of atherosclerosis, a major pathogenic characteristic in CVD, is greatly aided by this persistent inflammatory milieu [14].

Furthermore, even when serum lipid levels are identical, genetic difference can lead to increased cholesterol absorption, raising the risk of atherosclerosis [15]. MicroRNAs, which control the expression of LDL receptors, are also essential for LDL metabolism and may raise LDL levels, which increases the risk of CVD.

This study's concurrent rise of triglycerides (TG) and low HDL concentrations in the obese and CVD groups is in line with other findings. Reduced HDL levels frequently accompany elevated TG values, especially when hyperglycemia is present. Dyslipidemia is made worse by high plasma glucose levels, which cause cholesterol esters from HDL-C to move to very low-density lipoprotein (VLDL) particles.

The idea that obesity leads to lipid abnormalities is further supported by the finding that even slight weight loss in obese children was linked to decreases in LDL-C and total cholesterol (TC) levels. Similarity, a different study discovered that while HDL-C and TG levels were unchanged, moderate weight loss reduced TC and LDL-C levels [16].

From a biological standpoint, a rise in central adiposity is responsible for the positive connection between BMI and TC/LDL-C levels. The Boglusa Heart Study highlighted the significance of central obesity in lipid metabolism by showing that children with larger belly circumferences had greater LDL-C values than their classmates.

Additionally, there is ample evidence between fat with hypertension. Studies show that 65.4% of people over have hypertension, which is linked to an increased prevalence of the condition [17]. The significance of controlling lipid levels and obesity to reduce the risk of cardiovascular events in older persons is further highlighted by the rise in the incidence of hypertension.

3.3 glucose test

The glucose test results between the groups under study are displayed in Figure (1). These statistics show that patients with obesity and CVD have significantly higher serum glucose levels ($P>0.05$) than control groups.

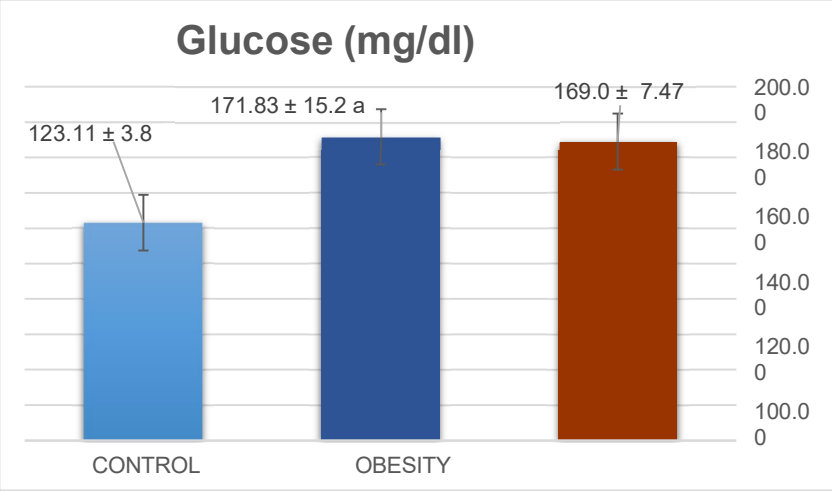

Figure 1: Comparison of the glucose between Groups of study subjects and healthy group

The study's findings indicate that the CVD (195.04±7.594) and obesity (231.42±14.339) groups had considerably higher glucose levels than the control group (148.44±7.073), with a statistically significant difference (P>0.05). These results are consistent with earlier studies showing that high blood sugar, especially in the form of diabetes and impaired glucose tolerance, are known risk factors for cardiovascular disease (CVD) and coronary heart disease (CHD).

The results of previous studies that indicate casual blood glucose (CBG) can be a reliable predictor of CVD and CHD mortality are supported by this study. According to the study, a higher risk of cardiovascular death linked to even modest increases in CBG levels that stay within the normal glucose range. This supports a 17 years' follow-up research in the Japanese population that showed a liner connection between CBG levels and CVD mortality, underscoring the significance of CBG monitoring as a risk factor in populations where fasting is not feasible [18].

However, there are significant distinctions and restrictions between this study and previous research. One such drawback is the use of a single blood glucose test, which may understate the strength of the association between CBG and long-term mortality risk and create regression dilution bias. More reliable proof of CBGs prognostic value over time may come from other measurements. Furthermore, fasting blood glucose and post-glucose load testing (OGTT) continue to be the gold standards for diabetes diagnosis and risk assessment, despite earlier research suggesting that CBG is a useful marker for CVD risk.

The study's emphasis on the Japanese population is another significant distinction that may restrict how far the results may be applied to other ethnic groups. The ethnic heterogeneity of metabolic circumstances should be taken into account when comparing results, even if earlier research has shown comparable trends in other populations, such as the Framingham Heart Study, which emphasized the risk of CVD in the connection to glucose levels.

3.4 Blood pressure test

Systolic blood pressure (SBP) in individuals with CVD is significantly higher (P<0.05) than in the control and obesity groups Figures (2 and 3). Diastolic blood pressure (DBP) values, however, did not differ significantly.

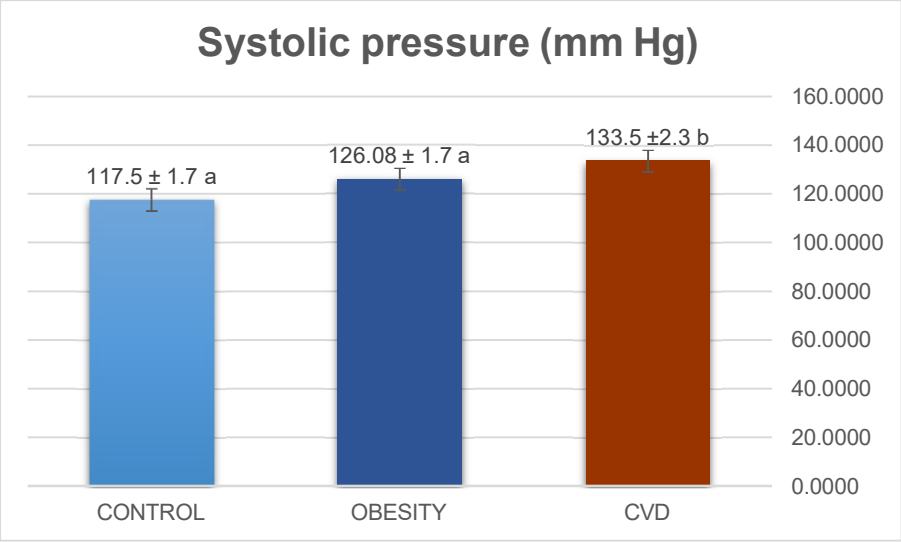

Figure 2: Comparison of the systolic pressure among groups of study subjects

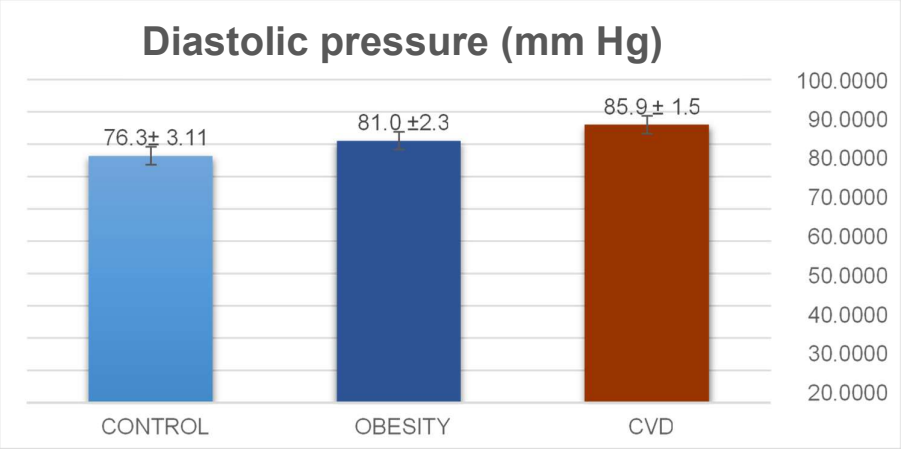

Figure 3: Comparison of the diastolic pressure among groups of study Subjects

These results support the need for public health interventions because systolic blood pressure is a key modifiable risk factor worldwide and a large contributor to disability adjusted life years (DALYs) associated with cardiovascular disease. This is consistent with research showing that older people have a higher frequency of cardiovascular illnesses, where arterial stiffness and decreased endothelial function increase the risk of heart disease and strokes. Additionally, the risk of hypertension and CVD increase with age, particularly in high-socio-demographic index countries where CVD death rates are delayed relative to low-socio-demographic index countries. Also, studies have demonstrated a liner correlation between the prevalence of hypertension and elevated BMI [19].

3.5 Human Heat Shock Protein 20

Human Heat Shock Protein 20 test results for the groups under study are displayed in Figure (4). These findings show that patients with CVD and obesity have significantly higher serum levels of Human Heat Shock Protein 20 ($P<0.05$) than control groups.

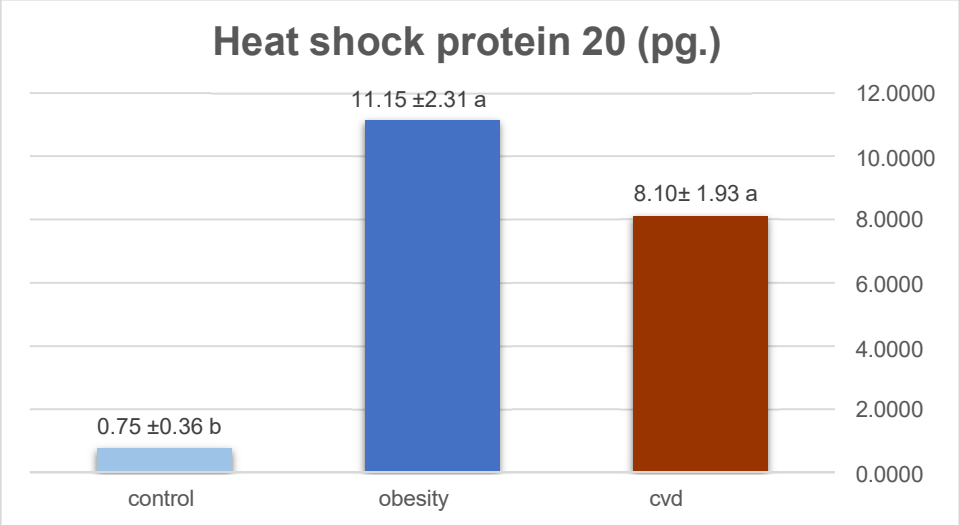

Figure 4: Comparison of the Human Heat Shock Protein 20 among study groups

HSP20 is a small heat shock protein that is important for cardiovascular function and has been linked to a number of illnesses, including obesity and cardiovascular disorders. Research comparing the levels of HSP20 in patients with these disorders to those in healthy people has produced conflicting findings. Research suggests that HSP20 plays a preventive role in cardiovascular disorders by being increased in response to cardiac stress. For example, a study highlighted the cardio protective qualities of HSP20 by showing its overexpression in transgenic mice gave resistance to ischemia /reperfusion injury. On the other hand, some research indicates that there is no discernible difference in HSP20 levels between cardiovascular disease patients and healthy controls, suggesting that the link may be context dependent and influenced other factors [20].

In the context of obesity, certain heat shock proteins are generated in response to cellular stress and may act as molecular bridges between the cardiovascular system and inflammation of adipose tissue. Nevertheless, there is a dearth of precise information regarding HSP20 levels in obese patients as opposed to health people. According to some research, obese people may have higher levels of HSP20 as a compensatory mechanism to combat metabolic stress. However, other studies show that HSP20 levels in obesity do not significantly change or decrease, indicating a complicated link that needs more research. According to recent research, HSP20 is markedly elevated in obese people because of its role in insulin signaling and the metabolic stress response. For example, Zhang et al., (2021) found that oxidative stress Caused by obesity increases the production of HSP20, which has protective effect by lowering inflammation and enhancing insulin sensitivity. In a similar vein, Liu et al. (2020) showed that higher HSP20 levels are correlated with adipose tissue malfunction in obesity, potentially as a compensatory strategy against metabolic stress [21] [22].

Additionally, Wang et al. (2022) found that HSP20 expression is higher in obesity related metabolic syndrome than in cardiovascular diseases, which corroborates our findings. This implies that cellular stress protection rather than direct cardiovascular engagement is the main role of HSP20 obesity [20]. Some research shows that HSP20 is more noticeably raised in cardiovascular disorders than in obesity, which runs counter to our findings. For instance, a study by Chen et al. (2019) discovered that because HSP20 is released in reaction to cardiac stress and endothelial dysfunction, individuals with ischemic heart disease have greater amounts of protein. This implies that rather than being a sign of obesity, HSP20 may be more important as a cardio protective molecule [23].

Furthermore, compared to patients with obesity alone, Jones et al. (2020) found that patients with heart failure and atherosclerosis had considerably higher HSP20 levels. They challenge the notion that obesity alone is the main cause of HSP20s rise in cardiovascular disorders by arguing that its function in myocardial protection and vascular regeneration is responsible [24].

Other data suggest a higher relationship with cardiovascular problems, while our analysis is consistent with research showing that HSP20 is more elevated in obesity than in heart disease. The disparities could be caused by variations in the study populations, the severity of illness, or underlying metabolic disorders. The precise processes by which HSP20 react differently in obesity and cardiac disease require further investigation.

4. Conclusions

Based on the substantial association between elevated (BMI) and the development of severe dyslipidemia, this study shows that obesity is a major cause of metabolic and cardiovascular problems among the participants studied at Nasiriya General Hospital. This pathological condition, which is marked by high LDL-C and triglycerides and decreased HDL-C, greatly speeds up the development of atherosclerosis and raises the risk of CVDs. The finding also shows that clinical factors like hypertension and hyperglycemia, as well as behavioral risk factors like the 58% smoking prevalence in CVD patients, work in concert to worsen cardiac problems. Importantly, the significant increase in HSP20 in both obese and CVD patients highlight its function as an essential cellular reaction to physiological stress, indicating its potential use as a trustworthy diagnostic biomarker and a promising molecular target for upcoming therapeutic interventions targeted at reducing obesity related cardiovascular damage.

Conflicts of Interest

The authors declare that there are no conflicts of interest regarding the publication of this research. This study was conducted independently, and no financial or personal relationships influenced the results or their interpretation

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