Investigation of Cardiac Damage and Coagulation Profile in Obese Holstein Cows

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SUMMARY

In parallel with the increase in the prevalence of obesity in humans in the world in recent years, this trend has also been observed in animals. Obese humans and pets are known to have myocardial damage. However, the presence of myocardial damage in cows that develop obesity naturally is not known. The aim of this study is to determine whether holstein obese cows have a myocardial damage and a coagulation disorder. This study consisted of 20 obese cows between -year-old 4-7, with an average weight of 856.6 kg, and 10 cows between the year-old 5-7, with an average weight of 650 kg. The concentrations of high-sensitivity Troponin I (hsTnI) ($p<0.002$), lactate dehydrogenase (LDH) ($p<0.001$), and creatine kinase (CK) ($p<0.002$) in the obese group were significantly higher compared to the control group. There was no significant difference between the obese and the control group levels of aspartate aminotransferase (AST) ($p>0.912$), creatine kinase-MB (CK-MB) isoform ($p>0.983$) and prothrombin time (PTsec) ($p>0.129$). The activated partial thromboplastin time (aPTT) concentration of the obese group ($p<0.028$) was found to be significantly lower than the control group. hsTnI concentration was found to have a positive correlation with AST ($p=0.002$; $r=0.538$), LDH ($p=0.001$; $r=0.813$), CK ($p=0.001$; $r=0.651$), white blood cell count (WBC) ($p=0.023$; $r=0.414$), lymphocyte count (LYM) ($p= 0.038$; $r=0.381$) and body condition score (BCS) ($p=0.017$; $r=0.431$).

In conclusion, in this study, we determined myocardial damage and coagulation disorders in obese Holstein cows. Additionally, significant changes in biochemical parameters were observed in obese Holstein cows. Future research should focus on determining whether myocardial damage in obese cows is reversible following weight loss and should include echocardiographic and electrocardiographic assessments for a more comprehensive understanding.

KEY WORDS

Cattle; Coagulation; hsTnI; Myocarditis; Obese.

INTRODUCTION

Obesity is an excessive increase in body fat as a result of the impairment of the body's energy mechanisms [1, 2]. Obesity arises from a complex interaction of various factors, including hormones that control hunger and fullness, different types of adipokines, cytokines, and the processes that generate heat in fat tissue. If any of these regulatory systems are disrupted, it can lead to an excessive buildup of energy in the body, ultimately resulting in obesity [2, 3].

Numerous investigations have demonstrated a noticeable rise in the occurrence of obesity among both humans and domestic animals [1,3]. The prevalence of obesity has been reported to be 34.1-40% in dogs, 11.5-35% in cats, and 24.5-45% in horses [4-10]. However, while there exists substantial data regarding the prevalence of obesity in pets and horses, the extent of research on the prevalence and systemic consequences of obesity in cattle remains unclear.

Obesity has been reported to cause significant damage to many different organs systemically in humans [1, 11, 12]. In a parallel context, it has been ascertained that obesity renders animals susceptible to an array of disorders encompassing orthopedic, respiratory, endocrine, urogenital, oncological, and dermal diseases [2, 4, 7]. In dairy cows exposed to experimentally induced obesity, there is a decrease in pregnancy rates and an increased need for intervention during calf birth, an increase in calf loss at birth and a decrease in milk production [13]. Besides, it has been reported that an increase in insulin resistance occurs in dairy cows that naturally develop obesity [14, 15]. Among the complications of obesity, the development of cardiovascular disorders holds a significant place [2, 16]. Obesity has several harmful impacts on the cardiovascular system. These effects result in higher systolic pressure in the pulmonary artery, an increase in the thickness of the cardiac walls, and a decrease in the cardiac ejection fraction, which is the percentage of blood the cardiac pumps out with each contraction [12, 16, 17]. Furthermore, reports have indicated that obesity contributes to the onset of coagulation disorders, encompassing both coagulation and fibrinolysis processes [12, 18]. Parameters like platelet count (PLT), prothrombin time (PT), and activated partial thromboplastin time (aPTT) are crucial for assessing the coagulation profile in ruminants. These parameters were evaluated to determine coagulation disorders that develop in diseases such as left displacement of the abomasum, ovario-

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hysterectomy, neonatal diarrhea and traumatic reticuloperitonitis [19-23].

Cardiac troponin I (cTnI) is a key protein in the complex interaction between actin and myosin, which are essential components for the cardiac muscle contractions and relaxations. It also plays a crucial role in regulating calcium levels at the point where actin and myosin interact [24, 25]. When the heart is injured, damaged muscle cells release troponins into the blood [25]. In veterinary medicine, cTnI is commonly used as a diagnostic marker for myocardial injury in conditions like abomasal displacement, peritonitis, metritis, mastitis, downer cow syndrome, uterine torsion, low calcium levels, and pericarditis [24, 26-28].

This study addresses a critical gap in the existing literature by investigating the impact of naturally developing obesity in Holstein dairy cows on their cardiovascular system, coagulation dynamics, and lipid structure. Our main objectives are as follows: i) To determine whether obese Holstein dairy cows show signs of myocardial impairment, we will measure specific cardiac biomarkers, including high-sensitivity cardiac troponin-I (hsTnI), creatine kinase (CK), creatine kinase myocardial band (CK-MB), lactate dehydrogenase (LDH), and aspartate aminotransferase (AST). ii) We will analyze the coagulation dynamics in these cows by assessing PT and aPTT. Additionally, we will explore potential associations between these coagulation parameters and the measured cardiac biomarkers. iii) To understand the relationship between body condition scores (BCS) and the lipid profile, which includes cholesterol, high-density lipoprotein (HDL), low-density lipoprotein (LDL), triglycerides, and very low-density lipoprotein (VLDL), in cows prone to obesity.

MATERIALS AND METHODS

Animal Selection

This was designed as a prospective study. Our study involved a group of 30 Holstein cows, split into two categories: 20 obese cows and 10 healthy cows. The obese cows were aged between 4 to 7 years, weighed an average of 856.6 kg, and had an average lactation number of 3.4. The healthy cows were 5 to 7 years old, with an average weight of 650 kg and an average lactation number of 2.2.

All cows came from the same farm and were kept in a semi-open barn that allowed for free movement. We assessed their body condition using a scoring system from 1 to 5, where a score of 4 or above indicated obesity. This scoring system was based on guidelines established by Edmonson et al. [29] and further defined by Kawashima et al. [30]. For the healthy group, cows with a Body Condition Score (BCS) between 2.75 and 3.5 were selected. In addition to these criteria, we conducted thorough clinical assessments of each cow, including checks of respiratory and heart rates, as well as body temperature. We excluded any cows that had respiratory, uterine, metabolic, or other health issues to maintain consistency and reliability in our comparison between the obese and healthy groups.

Laboratory Analyses

For hematological, biochemical and coagulation profile analyses, blood was taken from the animals in the study groups from the jugular vein in accordance with the technique. For haematological testing, we collected 3 ml of blood into tubes (BD Vacutainer®, Plymouth, UK) anticoagulated with K2E (EDTA). Similarly, for coagulation profile testing, 3 ml of blood was collected into sodium citrate tubes (Vacutte, sodium citrate 3.2%, Austria). For biochemical analyses, we drew 5 ml of blood into non-anticoagulant plain tubes (BD Vacutainer®, Plymouth, UK). After collection, the blood in the non-anticoagulant tubes was allowed to clot for about an hour at room temperature. We then centrifuged these samples at 5000 rpm for 5 minutes using a centrifuge (Hermle Z 36 HK®, Germany) to separate the serum. Coagulation profile analysis was analyzed by placing the blood in a refrigerator immediately after blood was taken and delivered to the laboratory within a maximum of 6 hours. We measured PT, aPTT, and PT SEC/sec using an automatic coagulation analyzer (Sysmex CS-5100, Siemens Healthcare Diagnostics, USA). Biochemical analyzes (LDH (U/L), CK (U/L), CK-MB (U/L), AST (U/L), ALT (U/L), GGT (U/L), cholesterol (mg/dL), HDL (mg/dL), LDL (mg/dL), VLDL (mg/dL), triglyceride (mg/dL), urea (mg/dL), and creatinine (mg/dL)) were used on an automated chemistry analyzer (Advia 1800, Siemens Healthcare Diagnostics, Malvern, PA, USA). hsTnI (ng/L) levels were determined using a Siemens Advia Centaur XPT kit, with these tests performed within one month of sample collection. The samples were stored at -20 oC until analyzed. We conducted comprehensive complete blood counts (including WBC ($x10^9$/L), LYM ($X10^9$/L), RBC ($X10^{12}$/L), HGB (g/dL), PLT ($x10^9$/L), PCT (%), and PDW (fL)) using a Mindray BC2800Vet® automated hematology device (Mindray Medical, China). Additionally, we measured blood beta-hydroxybutyric acid (BHBA) (mmol/L) levels using a FreeStyle Optimum Neo handheld device (Abbott Diabetes Care Ltd., Witney, UK) with specific test strips designed for these analyses.

Statistical Analysis

We analyzed the data using IBM SPSS Statistics version 26 (IBM Corp., Armonk, NY, USA) and GraphPad Prism version 9. Our descriptive statistics included the mean ± standard deviation, and the minimum and maximum values. To check if the data followed a normal distribution, we used the Shapiro-Wilk test. For comparing the obese and control groups of cows, we used different statistical tests based on the data distribution. If the data did not follow a normal distribution, we applied the Mann-Whitney U test. For data that did follow a normal distribution, we used the Independent Sample T test. To examine the relationships between various variables, we followed the approach outlined by Little and Hills [31] and used Spearman's rank correlation test. The interpretation of the correlation coefficients was as follows: $r > 0.60$ indicated a strong correlation, $0.30 < r < 0.60$ suggested a moderate correlation, and $r < 0.30$ implied a weak correlation. We set the statistical significance level at a p value of < 0.05 to determine significant differences between the groups.

RESULTS

The hsTnI ($p<0.002$), LDH ($p<0.001$) and CK ($p<0.002$) concentrations of the obese group were significantly higher than the control group. There was no significant difference in AST ($p>0.912$) and CK-MB ($p>0.983$) levels between the obese and control groups (Table 1).

The aPTT ($p<0.028$) concentration of the obese group was found to be significantly lower than the control group. There

Table 1 shows the mean±SD, min, max values of the cardiac values of obese and control group cows and the statistical differences between the groups.

Variables	Unit	Groups	Mean±S.D	Minimum	Maximum	P value*
hsTnI	ng/L	Obese Control	120.98±89.8 61.03±85.13	34.8 20.5	412 298.7	<0.002
LDH	U/L	Obese Control	1380.65±322.07 1049.4±173.08	820 855	1945 1402	<0.001
AST	U/L	Obese Control	102.75±39.57 94.7±20.28	57 72	185 140	>0.912
CK	U/L	Obese Control	422.25±314.93 180.8±83.81	136 103	1400 391	<0.002
CK-MB	U/L	Obese Control	201.9±81.96 203.69±75.9	90.8 94.8	349.40 394.70	>0.983

hsTnI, High-sensitivity cardiac troponin-I; CK, Creatine kinase; CK-MB, Creatine kinase myocardial band; LDH, Lactate dehydrogenase; AST, Aspartate aminotransferase. *p<0.001, p<0.005 is considered statistically significative.

is no significant difference between the PT (p>0.184) and PT-SEC (p>0.129) levels of obese and control group cows. LYM (p<0.002), PLT (p<0.002), PCT (p<0.011) and HGB (p<0.004) levels of the obese group were found to be significantly higher than the control group, but there was no difference in RBC (p>0.304) and WBC (p>0.180) values. The obese group was found to have significantly lower PDW (p<0.001) levels than the control group (Table 2).

Cholesterol (p<0.001), HDL (p<0.001), LDL (p<0.001) and urea (p<0.001) concentrations of the obese group were significantly higher than the control group. However, there were no significant statistical differences between the obese and control cows in terms of BHBA (p>0.934), triglyceride (p>0.198), VLDL (p>0.200), ALT (p>0.065), GGT (p>0.701) and creatinine (p>0.813) (Table 3).

The hsTnI showed significant positive correlations with AST (p = 0.002; r = 0.538), LDH (p=0.001; r =0.813), CK (p=0.001; r=0.651), WBC (p=0.023; r=0.414), LYM (p= 0.038; r=0.381),

Table 2 shows the mean±SD, min, max values of the coagulation and hematological profiles of obese and control group cows and the statistical differences between the groups.

Variables	Unit	Groups	Mean±S.D	Minimum	Maximum	P value*
PT	%	Obese Control	40.61±3.23 36.5±8.84	33.8 21.4	46.3 52.3	>0.184
PTSEC	Second	Obese Control	22.59±1.6 25.85±6.1	20.1 18.2	26.3 39.1	>0.129
APTT	Second	Obese Control	33.75±6.9 39.64±5.82	24.2 32.6	45.5 52.9	<0.028
WBC	$X10^9/L$	Obese Control	12.89±3.76 10.97±3.24	7 5.4	18.9 16.4	>0.180
LYM	$X10^9/L$	Obese Control	6.33±3.35 3.12±1.67	1.3 1.2	14.4 6.3	<0.002
RBC	$X10^{12}/L$	Obese Control	6.99±0.48 6.72±0.71	5.94 5.66	7.96 7.5	>0.304
PLT	$X10^9/L$	Obese Control	266.75±90.92 138.2±88.88	77 29	429 346	<0.002
PDW	fL	Obese Control	6.95±0.82 8.55±1.04	5.8 6.8	8.6 9.9	<0.001
PCT	%	Obese Control	0.13±0.05 0.08±0.05	0.04 0.01	0.21 0.17	<0.011
HGB	g/dL	Obese Control	10.68±0.66 9.49±1.12	9.90 7.80	12.10 11.10	<0.004

PT, prothrombin time; aPTT, active partial thromboplastin time; WBC, white blood cell; LYM, lymphocyte; RBC, red blood cell; PLT, thrombocyte; PDW, platelet distribution width; PCT, plateletcrit; HGB, hemoglobin. *p<0.001, p<0.005 is considered statistically significative.

Table 3 shows the mean±SD, min, max values of the biochemical and clinical findings of obese and control group cows and the statistical differences between the groups.

Variables	Unit	Groups	Mean±S.D	Minimum	Maximum	P value*
BHBA	mmol/L	Obese Control	0.36±0.17 0.35±0.13	0.1 0.2	0.8 0.5	>0.934
Cholesterol	mg/dL	Obese Control	171.90±45.29 97.05±21.04	102 71.4	293 139	<0.001
HDL	mg/dL	Obese Control	115.08±26.9 55.93±10.61	74.03 42.5	181.94 77.4	<0.001
LDL	mg/dL	Obese Control	44.27±14.25 22.34±9.38	18.9 7.4	80.2 36	<0.001
VLDL	mg/dL	Obese Control	4.68±1.48 3.94±1.42	2 1.3	7.6 6.2	>0.200
Triglyceride	mg/dL	Obese Control	23.4±7.38 19.67±7.14	10 6.2	38 31	>0.198
ALT	U/L	Obese Control	37.2±12.12 28±12.88	21 13	65 46	>0.065
GGT	U/L	Obese Control	25.8±6.4 24.9±5.07	13 19	39 32	>0.701
Urea	mg/dL	Obese Control	34.6±6.06 18.76±11.44	23 9.04	47 44	<0.001
Creatinine	mg/dL	Obese Control	0.89±0.15 0.88±0.18	0.64 0.6	1.27 1.26	>0.813

BHBA, beta hydroxy butyric acid; HDL, high-density lipoprotein; LDL, low-density lipoprotein; VLDL, very low-density lipoprotein; ALT, Alanine Aminotransferase; GGT, gamma glutamyl transferase. *p<0.001 is considered statistically significative.

and BCS (p=0.017; r=0.431), but had a significant negative correlation with PDW (p=0.005; r=-0.498). There is also a significant positive correlation between AST and LDH (p=0.001; r=0.682), CK (p=0.001; r=0.631), WBC (p=0.042; r=0.373), and ALT (p=0.010; r=0.465) levels. LDH shows a positive correlation with CK (p=0.001; r=0.702) and WBC (p=0.042; r=0.374), and a substantial negative correlation with PDW (p=0.005, r=-0.459). Similarly, CK correlates positively with BCS (p=0.016; r=0.435), cholesterol (p=0.008; r=0.473), HDL (p=0.011; r=0.459), LDL (p=0.023; r=0.415), and ALT (p=0.007; r=0.484), but negatively with PDW (p=0.006; r=-0.487). Furthermore, there is a significant positive correlation between BCS and LDL (p=0.001; r=0.608), HDL (p=0.001; r=0.760), and cholesterol (p=0.001; r=0.672) levels (Table 4).

While the BCS value of the obese group (p<0.001) was found to be significantly higher than the control group, the respiratory frequency (p<0.001) was found to be significantly lower (Table 5). hsTnI, PLT, aPTT and BCS values of the obese and control groups are presented in figure 1.

DISCUSSION

In many countries of the world, there is an important debate about whether obesity is a disease or not [11]. The core of these discussions is the critical role obesity plays in causing various health issues. This includes diabetes, heart diseases, joint problems like osteoarthritis, sleep apnea, asthma, and liver conditions not related to alcohol. Recent studies focusing on humans have already shown this connection [11, 32]. Unfortunately, there has been very little research into how obesity affects animals in veterinary medicine. However, drawing from what we know about obesity's impact on human health, some studies have looked into how it affects cats, dogs, and horses [2, 3, 8]. As of now, no research has specifically examined the link between natural obesity in Holstein cows and the related issues of myocardial injury and blood clotting disorders. The main goal of this study was to detect heart muscle damage in obese Holstein cows by examining specific cardiac biomarkers (like hsTnI, LDH, CK, CK-MB, and AST). We also aimed to outline their blood clotting profile (including PT, PLT, PTSEC, and APTT) and investigate any potential clotting issues. Additionally, the research sought to clarify how obesity affects blood and biochemical parameters in this group of cows. In this study, the concentration of hsTnI, CK and LDH in obese Holstein cows was significantly higher than in the control group, suggesting a myocardial injury. In addition, the fact that the aPTT level is significantly lower and the PLT number is significantly increased in obese cows suggesting that there is a coagulation disorder. There is a significant correlation between the BCS value and hsTnI, HDL, LDL and cholesterol levels in obese cows. This current study is the first study to investigate myocardial damage, coagulation disorder and haemato-biochemical values in obese Holstein cows.

In recent years, there's been a noticeable parallel trend: as human obesity rates rise, a similar increase is seen in animal pop-

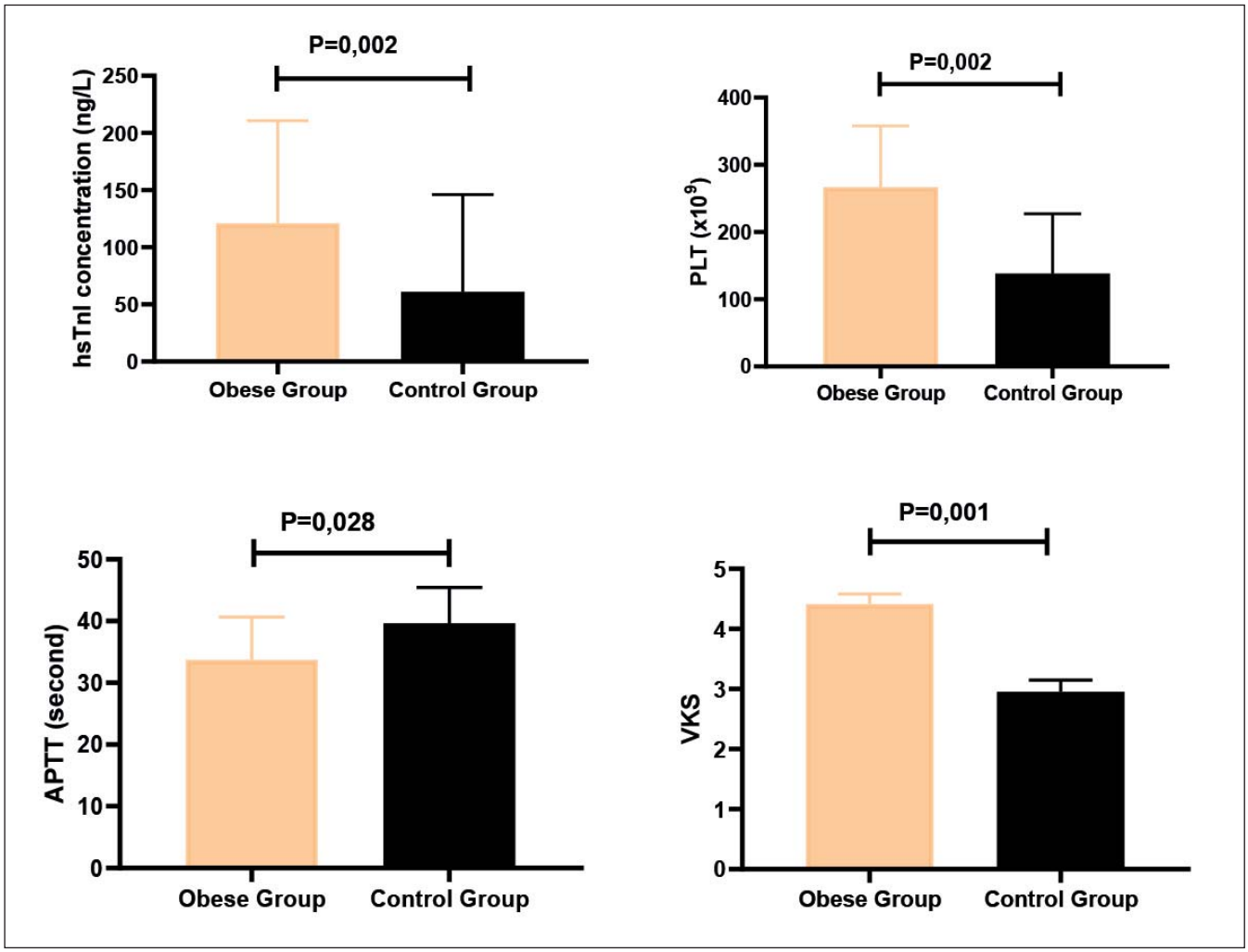

Figure 1 shows hsTnI, PLT, aPTT and BCS values of obese and control group cows.

hsTnI, High-sensitivity cardiac troponin-I; PLT, trombosit; aPTT, active partial thromboplastin time; BCS, body condition score. p<0.001, p<0.005 is considered statistically significative.

Table 4 shows the Spearman correlation analysis between cardiac and biochemical parameters in the study.

Variables	hsTn1	AST	LDH	CK	BCS
AST	0.538*				
LDH	0.813**	0.682**			
CK	0.651**	0.631**	0.702**		
WBC	0.414*	0.373*	0.374*		
LYM	0.381*				
PDW	0,-498*		0,-459*	0,-487*	
BCS	0.431*			0.435*	
Cholesterol				0.473*	0.672**
HDL				0.459*	0.760**
LDL				0.415*	0.608**

hsTnI, High-sensitivity cardiac troponin-I; CK, Creatine kinase; CK-MB, Creatine kinase myocardial band; LDH, Lactate dehydrogenase; AST, Aspartate aminotransferase; BCS, body condition score; HDL, high-density lipoprotein; LDL, low-density lipoprotein; WBC, white blood cell; LYM, lymphocyte; PDW, platelet distribution width.
*Correlation is significant at the p< 0.05 level. **Correlation is significant at the p< 0.01 level.

Table 5 shows the clinical parameters of obese and control group cows and statistical differences between groups.

Clinical parameters	Unit	Groups	Mean±S.D	Minimum	Maximum	P value *
Heart Rate	beats per minute	Obese Control	80.4±16.15 78.6±15.2	56 56	112 108	>0.772
Respiration Rate	breaths per minute	Obese Control	24.2±3.99 31.6±6.65	20 24	32 48	<0.001
Body Temperature	°C	Obese Control	38.3±0.26 38.44±0.3	37.9 38	38.8 38.9	>0.202
BCS		Obese Control	4.41±0.17 2.95±0.2	4 2.75	4.75 3.25	<0.001
Lactation number		Obese Control	3.4±0.8 2.2±1.4	4 2	9 6	0.025
Weight	Kilogram	Obese Control	856.6±0.2 650±0.8	650 550	1000 700	0.010

BCS, body condition score. *p<0.001, p<0.005 is considered statistically significative

ulations [33, 34]. Obesity leads to significant effects on adipose tissue and also disrupts the complex balance of hemostasis, coagulation, and fibrinolysis processes [18, 35]. The effects of having more fat around internal organs greatly influence blood clotting and the breakdown of clots. This happens because the fat tissue produces and releases a substance that can slow down the process of breaking down clots. Indirectly, substances produced by fat tissue, like leptin and adiponectin, play a role. They activate platelets in the blood, creating an environment that promotes clot formation and slows down the breakdown of these clots [12, 18]. In studies conducted on obese people, it has been reported that coagulation disorders occur [36]. Similar to these findings, a study by Bilge et al. [37] on obesity in children showed notable decreases in the time it takes for blood to clot and an increase in the number of platelets, which are critical for clotting. Likewise, in a study conducted on obese and overweight dogs, it was reported that the aPTT duration was significantly reduced and the PLT number was significantly increased [38]. In a related study by Kaji et al. [35] involving rats, there was a notable shortening in the time it takes for blood to clot. Consequently, the use of a shorter aPTT time as a diagnostic tool has gained significant attention in various research studies [35, 38]. Additionally, the studies undertaken by Pasquini et al. [39] on obese dog and Duburcq et al. [12] on obese swine corroborated a marked increase in PLT count. In this study, examining obese cows revealed a notably shorter aPTT time compared to the control group, although no significant differences were found in other blood clotting proteins. At the same time, the counts of PLT and LYM were much higher in obese cows. Taken together, these findings suggest that obesity might create conditions that promote inflammation and an increased tendency for blood clotting.

Obesity has a deep effect on many organs in the body, especially the heart and blood vessels, causing a range of disorders through both direct and indirect impacts. The direct effect includes changes in the structure and function of the cardiovascular system due to excess body weight. The indirect effect involves triggering risk factors like insulin resistance, high blood sugar, and high blood pressure, which can lead to heart diseases [18, 40]. Echocardiographic studies investigating how obesity affects dogs' hearts have uncovered several changes. These include enlargement of the left ventricle, reduced ability of the heart to relax between beats, increased systolic blood pressure, thickening of the wall between the heart's chambers, an imbalance in the heart's response to sympathetic and vagal nerve signals, and a decrease in the heart's ability to pump blood efficiently [17, 41-43]. On the other hand, postmortem studies looking into the cellular and tissue changes due to obesity in horses have revealed specific heart-related issues. These include abnormalities in the structure of the aorta's wall, cell vacuolization, and thickening of the inner layer of the pulmonary and coronary arteries [44, 45]. In recent years, the development of heart-specific biomarkers has become crucial for early detection of myocardial injury. hsTnI is one such biomarker, notable for its exceptional ability to detect very low concentrations. This makes it more sensitive than the traditionally used cTnI [46, 47]. cTnI is particularly important because it is specific to the heart, making it an ideal marker for indicating heart muscle damage [25]. Lyngbakken et al. [48] reported increased levels of hsTnI in obese individuals, which decreased after bariatric surgery. Supporting these observations, another study with 9.739 participants revealed a higher risk of subclinical heart muscle damage in individuals who have been obese for their entire lives [49]. Similar results appear in veterinary research. Aptekman et al. [50] and Cihan and Tural [51], in their studies on obese dogs, found higher levels of cTnI. In similar research, other researchers have observed that cTnI levels significantly increase in obese dog but normalize after weight loss [34]. In our study, we found that hsTnI levels were significantly higher in obese cows compared to the control group, which is consistent with findings from other studies [49-51]. The strong link between BCS value and hsTnI level in obese Holstein cows is particularly notable, suggesting cardiovascular damage. Increased hsTnI levels suggesting of cardiac muscle damage in this study may be related to disruptions in lipid metabolism, changes in the coagulation profile, and altered inflammatory status.

Serum levels of CK and LDH enzymes are widely used as indicators to identify both normal and abnormal conditions of skeletal muscle tissue [25-52]. An elevation in the levels of these enzymes serves as a discerning marker of tissue damage and cellular necrosis [53, 54]. Azza et al. [52] carried out a study on obese male rats, where they observed a significant increase

in the activities of CK and LDH. In our current study, we found noticeable differences: the concentrations of LDH and CK enzymes in obese cows were substantially higher than in the control group. Notably, there was a positive correlation between LDH concentration and both CK and WBC. Additionally, CK showed positive correlations with BCS, cholesterol, HDL, LDL, and ALT. This increase in enzyme concentrations probably indicates the development of cardiac damage, and when evaluated together with cardiac-specific hsTnI, it is also an important finding supporting the existence of myocardial damage. Adipose tissue plays a key role in controlling energy metabolism by releasing adipokines, which are substances that regulate how energy is stored and distributed in the body. Additionally, the hormone-producing function of adipose tissue affects many different body processes, including the metabolism of fats and the expenditure of energy [55]. Imbalances in adipose tissue can lead to disturbances in fat metabolism, resulting in the development of obesity [56]. The complex relationship between obesity and lipid profiles has been confirmed in a range of studies involving both humans and companion animals [56, 57]. A study involving obese children showed a significant increase in cholesterol and LDL levels. This was accompanied by echocardiographic evidence of impaired heart function. The findings included increased left ventricular fractional shortening, greater left ventricular mass, and a higher left ventricular mass index [57]. Cihan and Tural [51] found significant rises in cholesterol and triglyceride levels in obese dogs, and they established a meaningful link between these factors and cTnI concentrations. Another study examined the effect of obesity on lipid profiles and heart function in dogs through echocardiography [42]. In line with previous research, our current study shows higher levels of cholesterol, HDL, and LDL in the obese group compared to the control group. Although the obese group had increased triglyceride and VLDL levels, there wasn't a significant difference between the two groups. We also found a strong correlation between the BCS value and HDL, LDL, and cholesterol. These results align with earlier studies on obesity [42, 51, 57], and suggest a major disruption in the metabolism of adipose tissue, leading to significant changes in the lipid profile.

This study has some limitations. No turbulence, murmur or pathological sound was detected in the cardiac examination performed on obese cows. However, due to the living space of the animals in this study and difficulties in obtaining materials, echocardiography and thorax radiography examination could not be performed.

CONCLUSION

As a result, in this study, cardiac and coagulation parameters in obese Holstein cows changed significantly, indicating that myocardial damage and coagulation disorders occur. In future studies on obese cows, it is necessary to determine whether myocardial damage after weight loss is reversible and to perform a complete cardiological evaluation.

Ethics approval

The execution of this current research study transpired subsequent to obtaining the requisite endorsement from the Bingöl University Animal Experiments Local Ethics Committee (B.U AELEC), as substantiated by the official documentation bearing the date 2023/04 and denoted as Decision No: 04/01.

Conflict of Interest Statement

The author declares that there is no conflict of interest.

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Data availability

The datasets generated during and/or analyzed during study are available from the corresponding author on reasonable request.

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