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Research Article

The Influence of Body Weight on Cholelithiasis among a Group of Kurdish Women in Garmian Province, Kurdistan Region of Iraq

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ABSTRACT

The existence of amounts of cholesterol or bilirubin in the bile can develop solid particles that precipitate in the gallbladder or biliary tract, which can become gallstones (GSs). The aim of the recent study is to investigate which anthropometric obesity indicator among the several ones is the most effective at predicting GS disease in Gallstone disease (GSD) depending on body weight. The study included 161 female subjects aged 20–70 years. Eighty-one of them had GSs, and eighty adult healthy females were also enrolled. All of them had been diagnosed with ultrasonography for GS detection. Also, they were divided into two subgroups according to body weight and age. The data were analyzed using unpaired t-tests in the Statistical Package for Social Sciences program; a value of $P < 0.05$ was considered statistically significant. The result of the group body mass index (BMI) <30 showed that the mean values of systolic blood pressure, diastolic blood pressure, fasting blood sugar (FBS), Total serum bilirubin (TSB), and low-density lipoprotein (LDL) were significantly higher and levels of albumin and high-density lipoprotein were significantly lower in the GSD group compared with the healthy group. The results of the group BMI >30 showed that the mean values of FBS, TSB, alanine aminotransferase, aspartate aminotransferase, LDL, and Very-low-density lipoprotein were significantly higher in the GSD group compared with the control group. We concluded that body weight is associated with GSs formation.

Keywords: Cholelithiasis, gallstone, cholesterol, gallbladder, body weight

INTRODUCTION

Gallstones (GSs) can form in the gallbladder or biliary tract due to abnormally high levels of bilirubin or cholesterol in the bile.[1] GSs affect between 10% and 20% of adults worldwide, making it a moderately common condition.[2] Health insurance should cover a large portion of the cost of hospitalization and therapy for Gallstone disease (GSD) individuals, even though the death rate is less.[3]

Laparoscopic surgery was utilized in around 90% of instances for cholecystectomy, the most typical surgical technique for treating GSD and related problems worldwide.[2] Cholecystectomy is a treatment option for risk factors for GSD, including GS pancreatitis, symptomatic cholelithiasis, acalculous cholecystitis, biliary dyskinesia, acute or chronic cholecystitis, and gallbladder tumors or polyps.[4] As well as metabolic syndrome and heart diseases, type 2 diabetes, and high blood pressure. Numerous variables, such as age, gender, body mass index (BMI), type 2 diabetes, non-alcoholic fatty liver, and insulin resistance, have been related to GSs as a risk factor.[5,6] GSs are more common in obese people.[7] It can be categorized as cholesterol, pigment, or mixed GSs depending on the main biochemical components of each type.[8]

The BMI is the most commonly used indicator of overall body fatness.[9] Without considering the distribution of fat in the abdomen, a number of studies point to the role of central obesity, a major factor in the progress of metabolic syndrome, in the development of GSs.[10] Cholesterol metabolism products and supersaturation of the bile ducts cause cholesterol-related stones to occur in over 90% of people, fat or not.[11]

Sometimes GS production is caused by increasing body weight, the degree of a person's obesity can be determined by using BMI, abdominal parameters and the ratio of waist-to-hip. Later, central abdominal fat is linked to insulin resistance,

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followed by an elevation in hepatic cholesterol release, regarded as one of the different mechanisms participating in the production of GS.[12] Obese individuals are more likely to get acute pancreatitis and have more severe symptoms as a result of several pathogenic variables, such as visceral obesity, supersaturated bile, and crystal formation.[13] According to several studies, GSs have been related to age, gender, BMI, non-alcoholic fatty liver, type 2 diabetes, and insulin resistance.[5,14] Another study concluded that GSs are more common in obese individuals.[15] Based on a study, females are more prone to develop GS formation, especially during gestation and when using contraceptives.[2] Zhang *et al.* demonstrated that both general obesity and central obesity are independent risk factors for GS disease. There finding suggest that abdominal fat plays a key role in metabolic changes that promote GS formation.[16] In addition, Chen *et al*. 2025 reported than indivadual with long-term obesity or significant weight gain during adulthood have ahigher risk of developing GSs later in life. However, rapid weight loss has also been identified as a risk factor due to increased cholesterol mobilization and bile supersaturation.[17] Through mechanisms involving cholesterol metabolism, bile composition, and gallbladder function, the literature consistently demonstrates that increased body weight is a substantial risk factor for cholelithiasis. Therefore, the main aim of this study was to investigate the association between GS formation and obesity in female Kurdish adults.

Study Population

A case–control study design was used. Information was gathered on patients who came to the Garmian Medical Centre in Kalar, Sulaymnia, Kurdistan, Iraq. Once every patient had given their permission to take part in this study, every person was contacted after the hospital's clinical diagnosis based on the clinical examination.

Eighty-one female groups, both with symptomatic and asymptomatic, GS disease, and eighty free of GS regarded as controls. All were diagnosed using ultrasonography; according to BMI, they were divided into two subgroups: BMI more than 30 and BMI <30. In order to compare the biochemical markers linked to GS, an unpaired t-test was used. All of them visited the hospital between June 2021 and October 2022.

Measurements

Using a conventional formula, BMI, waist circumference (WC), waist-to-height ratio, and waist-to-hip ratio (WHR) were determined. Furthermore, measurements of the diastolic and systolic blood pressure (SBP) were made. Every patient gave their informed consent to take part in the trial, and everyone knew what the goals of the study were. The University of Garmian's ethical committee (Ethical permission No. 7: July 20, 2021) monitored the study's execution, which adhered to the Helsinki Declaration.

Laboratory Investigations

From each participant had 5 mL of blood was collected. Then treated with a gel tube containing clotting-activated factor, the serum was separated and preserved at −86 until biochemical analysis. The levels of (lipid profiles and enzymatic and non-enzymatic liver functions) were evaluated using (Cobas c 111-Roch) an automatic biochemical analyzer. The levels of adiponectin and visfatin were estimated using (Thermo Fisher USA) Eliza reader and washer at the biology lab, Garmian University.

Statistical Analysis

For analysis of data, IBM's Statistical Package for Social Sciences software, version 21.0 (IBM, Chicago, IL, USA) was used. To compare between case and control, an unpaired t-test was used, and the result was presented as mean ± standard error of the mean. $P < 0.05$ was considered significant.

RESULTS

In the present study the results showed, the ratio of normal weight and obese individuals in Figure 1 and the gall stone picture with gall bladder after surgery were found in Figure 2, and the mean value of SBP, diastolic blood pressure (DBP), fasting blood sugar (FBS), total serum bilirubin (TSB), and low-density lipoprotein (LDL), were significantly higher and levels of albumin and high-density lipoprotein (HDL) were remarkably lower in GSD group compared with healthy group. While other parameters did not change significantly [Table 1].

The results in Table 2 showed the mean levels of FBS, TSB, ALT, AST, LDL, and VLDL, were higher significantly in GSD group compared with control group.

According to multivariate logistic regression analysis: increased WT, BMI, LDL, and Amylase whereas the age, WHR, triglyceride (TG), TC, HDL, and VLDL were not identified as significant risks for the development of GSs as shown in [Table 3].

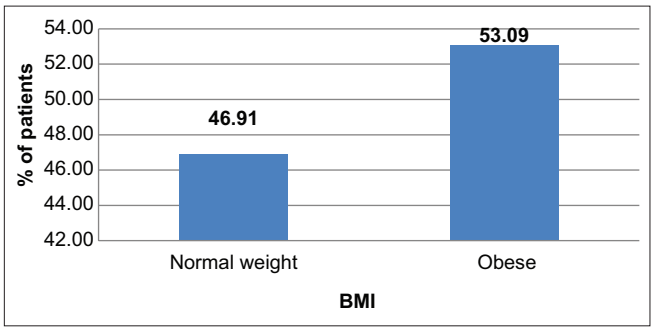

Figure 1: The percentage of obese and normal weight in gall stone patients

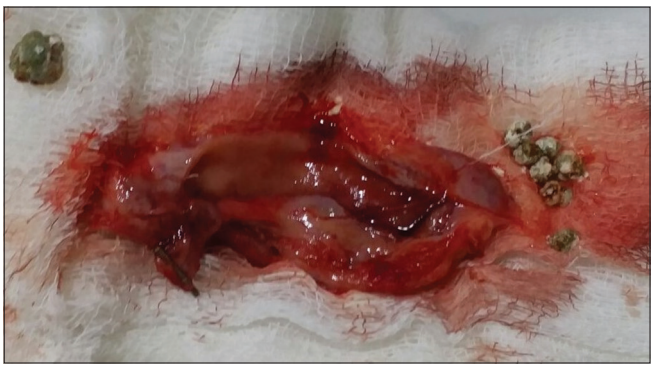

Figure 2: Gall bladder with gallstone after removing

Table 1: Clinical and biochemical parameters in case and control BMI <30

Parameters	Control group BMI <30 (Mean±SE)	Gallstones group BMI <30 (Mean±SE)	*P*-value
BMI (kg/m^2)	25.72±0.36	26.10±0.37	0.493
Age (years)	42.68±1.33	45.89±2.39	0.245
WC (cm)	88.20±1.61	89.82±2.00	0.533
WHTR (%)	0.54±0.06	0.56±0.05	0.098
WHR (%)	0.91±0.11	0.91±0.01	0.098
SBP (mm/Hg)	114.91±2.28	126.64±3.05	0.007*
DBP (mm/Hg)	76.33±1.41	83.61±2.14	0.014*
FBS (mg/dL)	95.36±2.32	117.34±3.93	0.001*
WBC (cell/mm^3)	7.26±0.41	8.32±0.44	0.143
TSB (Mg/dl)	0.48±0.04	0.68±0.07	0.021*
ALT (U/L)	18.95±2.34	25.11±3.87	0.184
ASP (U/L)	19.43±1.19	20.73±1.12	0.430
ALP (U/L)	70.66±4.08	72.66±6.49	0.798
Visfatin (ng/mL)	2.23±0.42	2.36±0.50	0.850
Adiponectin (mg/L)	7.08±0.85	5.75±0.62	0.227
Amylase (U/L)	53.33±2.61	62.51±4.69	0.074
Lipase (U/L)	26.80±1.55	32.91±4.15	0.181
TG (mg/dL)	118.67±7.81	133.09±9.47	0.250
TC (mg/dL)	167.78±4.32	186.80±8.50	0.053
HDL (mg/dL)	44.70±2.23	38.40±1.33	0.020*
LDL (mg/dL)	95.38±4.36	139.34±13.19	0.003*
VLDL (mg/dL)	34.10±1.00	37.49±2.39	0.199
Albumin (g/dL)	4.91±0.12	4.07±0.10	0.001*
TP (g/dL)	6.43±0.21	6.80±0.29	0.308

BMI: Body mass index, WC: Waist circumference, WHTR: Waist-to-height ratio, WHR: Waist-to-hip ratio, SBP: Systolic blood pressure, DBP: Diastolic blood pressure, FBS: Fasting blood sugar, WBC: White blood cells, TSB: Total serum bilirubin, ALT: Alanine aminotransferases: Aspartate aminotransferase, ALP: Alkaline phosphatase, TG: Triglyceride, TC: Total cholesterol, HDL: High-density lipoprotein, LDL: Low-density lipoprotein, VLDL: Very-low-density lipoprotein, TP: Total proteins. At $P<0.05$, independent T-tests were employed

Table 2: Clinical and biochemical parameters in case and control BMl >30

Parameters	Control group BMI >30 (Mean±SE)	Gallstones group BMI >30 (Mean±SE)	*P*-value
BMI (kg/m^2)	34.06±0.52	34.26±1.03	0.843
Age (years)	44.43±2.47	45.00±1.77	0.853
WC (cm)	106.05±2.78	108.86±2.14	0.438
WHTR (%)	0.66±0.017	0.65±0.02	0.830
WHR (%)	0.91±0.013	0.93±0.040	0.293
SBP (mm/Hg)	120.6±3.4	128.3±2.4	0.140
DBP (mm/Hg)	83.90±2.89	83.34±1.62	0.871
FBS (mg/dL)	99.83±3.86	129.92±6.94	0.015*
WBC (cell/mm^3)	6.77±0.46	8.71±0.62	0.066
TSB (Mg/dL)	0.42±0.07	0.67±0.07	0.024
ALT (U/L)	19.05±3.42	33.70±4.51	0.035*
AST (U/L)	18.24±1.47	35.90±7.28	0.023*
ALP (U/L)	82.73±6.97	82.62±9.06	0.995
Visfatin (ng/mL)	2.04±0.46	1.66±0.21	0.411
Adiponectin (mg/L)	8.22±1.77	5.57±0.33	0.055
Amylase (U/L)	56.46±3.57	60.80±5.08	0.493
Lipase (U/L)	24.80±6.55	22.90±1.13	0.630
TG (mg/dL)	123.86±7.46	139.06±15.65	0.335
TC (mg/dL)	178.00±7.09	195.81±5.85	0.060
HDL (mg/dL)	42.86±5.20	40.01±1.34	0.483
LDL (mg/dL)	102.91±11.10	144.12±7.51	0.049*
VLDL (mg/dL)	35.60±1.17	40.10±1.75	0.038*
Albumin (g/dL)	4.50±0.09	4.33±0.21	0.427
TP (g/dL)	6.74±0.26	7.27±0.31	0.268

BMI: Body mass index, WC: Waist circumference, WHTR: Waist-to-height ratio, WHR: Waist-to-hip ratio, SBP: Systolic blood pressure, DBP: Diastolic blood pressure, FBS: Fasting blood sugar, WBC: White blood cells, TSB: Total serum bilirubin, ALT: Alanine aminotransferases: Aspartate aminotransferase, ALP: Alkaline phosphatase, TG: Triglyceride, TC: Total cholesterol, HDL: High-density lipoprotein, LDL: Low-density lipoprotein, VLDL: Very-low-density lipoprotein, TP: Total proteins. At $P<0.05$, independent T-tests were employed

The findings of the univariate logistic regression analysis display that age, education levels (Secondary school and Institute), marital status, pregnancy (uniparous), WT, Waist/Hip, TG, visfatin, were not associated with the incidence of GS disease. Whereas, education levels (Illiterate and primary school), Pregnancy (multiparous) and physical activity were associated with GS occurrence.

Among the continues variables, the result noted that BMI, HDL, LDL, VLDL, Adiponectin, SBP, DBP, FBG and ALT were associated with the risk of developing GSs [Table 4].

DISCUSSION

Because GSs are more common in women, only women were included in the current study. According to the results of a related study, women are more prone to have GSs.[18]

BMI <30 the mean value of SBP, DBP, FBS, TSB, and LDL, were significantly higher and levels of albumin and HDL were remarkably lower in GSD group compared with healthy group.

When comparing cases to controls according to BMI and age we found that blood pressure in patients significantly increased. a study,concluded patients with GSD and cholelithiasis had greater SBP and DBP than healthy people.[19] GSs and cardiovascular disease may be related because of the same pathophysiology of metabolic pathways involved in the metabolism of cholesterol. One cardiovascular condition that can lead to cholesterol accumulation in blood arteries is hypertension. One of the main characteristics of GSs and atherosclerosis is the buildup of cholesterol. The development of blood vessel atherosclerosis can raise blood pressure, which in turn raises the risk of cardiovascular disease and, indirectly,

Table 3: Risk factors associated with gallstone disease in multivariate logistic regression

Variable	B	OR	95% CI	*P*-value
Age	−0.013	0.987	0.950–1.026	0.522
WHR	1.143	3.135	0.001–158.03	0.792
WT	0.084	1.088	1.059–1.186	0.013*
BMI	0.289	1.335	1.103–1.614	0.003**
TG	0.008	1.008	0.994–1.022	0.261
TC	−0.006	0.994	0.974–1.015	0.580
HDL	−0.042	0.959	0.911–1.009	0.107
VLDL	−0.023	0.977	0.883–1.081	0.650
LDL	0.053	1.054	1.029–1.079	0.007**
Amylase	0.059	1.061	1.024–1.098	0.001*

WHR: Waist-to-hip ratio, WT: Weight, BMI: Body mass index, TG: Triglyceride, TC: Total cholesterol, HDL: High-density lipoprotein, VLDL: Very low-density lipoproteins, SE: Standard error, *Significant $P<0.05$, ** Highly significant $P<0.01$

Table 4: Risk factors associated with gallstone disease in univariate logistic regression

Variable	B	OR	95% CI	*P*-value
Age	0.16	1.017	0.990–1.044	0.225
Education levels (University)				
Illiterate	1.013	2.755	1.014–7.489	0.047*
Primary school	1.761	5.816	0.627–8.556	0.006**
Secondary school	0.203	1.224	0.267–5.605	0.794
Institute	−0.219	0.804	0.219–2.943	0.741
Marital status	0.412	1.510	0.545–4.187	0.428
Pregnancy (nulliparous)				
Uniparous	0.513	1.670	0.479–5.827	0.421
Multiparous	1.284	3.609	1.475–8.835	0.005**
Physical activity	2.112	8.268	3.927–17.408	0.001**
WT	0.021	1.021	0.999–1.044	0.061
Waist/Hip	1.320	3.743	0.006–253.483	0.691
BMI	0.101	1.107	1.035–1.184	0.003*
TG	0.009	1.009	0.999–1.018	0.073
HDL	−0.056	0.946	0.914–0.979	0.002*
LDL	0 0.041	1.041	1.026–1.057	0.001*
VLDL	0.080	1.083	1.033–1.136	0.003*
Amylase	0.47	1.048	1.021–1.076	0.001*
Visfatin	0.041	1.042	0.833–1.305	0.717
Adiponectin	0.148	1.160	1.002–1.343	0.047*
SBP	0.055	1.057	1.020–1.095	0.002*
DBP	0.048	1.049	1.003–1.097	0.036*
FBG	0.072	1.075	1.040–1.111	0.041*
ALT	0.059	1.061	1.013–1.112	0.013*

WT: Weight, BMI: Body mass index, TG: Triglyceride, HDL: High-density lipoprotein, LDL: Low-density lipoproteins, VLDL: Very low-density lipoproteins, SBP: Systolic blood pressure, DBP: Diastolic blood pressure, FBG: Fasting blood sugar, ALT: Alanine transaminase, SE: Standard error, *Significant $P<0.05$, ** Highly significant $P<0.01$

GS disease.[20] Another study confermed thres no significance defference in blood presure between GSD and healthy.[21]

BMl >30 the results in Table 2 showed the mean levels of FBS, TSB, ALT, AST, LDL, and VLDL, were higher significantly in GSD group compared with control group.

According to BMI the mean value of FBS, TSB, LDL were significantly higher and levels of albumin were significantly lower in GSD group compared with healthy group.

The current study's findings on blood glucose revealed that GS patients had statistically significant elevations in their glucose levels. The findings were consistent with the majority of earlier research, which shown a high level of GSD in individuals with diabetes.[22] GS prevalence in diabetes patients may be higher due to a number of factors, including biliary supersaturation, modifications in cholesterol nucleation, and reduced gallbladder motility. Patients with GSs had considerably lower serum albumin levels, and comparable findings revealed a similar association to the one identified in the current investigation.[23]

In both group which BMI <30 in the present study the results showed HDL were significantly lower in GSD group compared with healthy group. It was discovered that having high HDL serum did not promote the development of GSs. Low HDL levels have been linked to GS formation, according to a different study by Serin *et al*.[21] Moreover, research conducted has demonstrated that HDL serves as a protective factor against GSs while TC, TG, and LDL levels are risk factors.[24]

Because of the elevated level of cholesterol secreted by the liver, obesity is an established risk factor for the formation of GSs.[12] There has been increasing interest in the relationship between central obesity indexes and clinical signs of metabolic syndrome.[25] Additionally, there is a strong correlation between insulin resistance, diabetes mellitus, and abdominal (central) obesity and GS formation.[26] While BMI is a measure of lean body mass, it is also the most widely used technique for assessing adiposity.[10] More specifically, central obesity is associated with the waist-to-hip, waist-to-height, and WC ratios. With varying degrees of success, several researches have looked into the relationship between these central adiposity indices and GSD.

In group which BMI >30 Individuals who have increased ALT levels may have aberrant liver function, which reflects the liver's health. According to the study's findings, there is a statistically significant positive correlation between GSD patients' serum total bilirubin levels, ALT, and AST. The outcome was consistent with a study that found a link between the level of liver function enzymes and GSD patients.[27]

Although there was a non-significant correlation between the formation of GSs and serum ALP levels, the results aligned with previous research indicating that individuals with GSD typically had elevated serum ALP levels. However, the association did not prove a statistically significant link between the high levels of this enzyme and GS formation.[28] Patients with GSs had considerably lower serum albumin levels, and comparable findings revealed a similar association to the one identified in the current investigation.[23]

We observed that there was no discernible change in the (BMI). Numerous studies show that those who are the most

obese have a significantly increased relative risk for GSD.[12] Reduced gallbladder contractility with meals and cholesterol supersaturation in the bile are linked to obesity, and both conditions may cause lithogenesis.[29]

There was no obvious distinction in the WHR between the patients and the controls. Still, Balakrishnan *et al*.,[19] have documented a rise in the patients' WHR as compared to the control group. This is because our research solely included female participants, whereas research involving both genders may result in disparities in the findings. When comparing cases to controls, we found that blood pressure in patients significantly increased. According to a study, patients with GS disease and cholelithiasis had greater SBP and DBP than controls.[19]

Adiponectin, which is released by fat cells, has another connection to the development of GSs. Obesity can stimulate the excretion of extra cholesterol accumulated in adipose tissue by regulating biliary lipid metabolism and raising leptin secretion. Consequently, elevated serum leptin levels can raise the release of cholesterol into bile, which can lead to supersaturation of bile with cholesterol and a higher risk of GSs.[30] Also, Obesity can lower serum adiponectin, which can cause insulin resistance. Insulin resistance can then result in a number of metabolic problems, including an increased risk of GSs.[31] The limitation and recommendations of the study, our study only included females, but the results warrant additional analysis with a larger sample size. We suggest doing another study on GSs that looks at the genetic factors that affect GS production.

CONCLUSION

In both group which BMI <30 In the present study the results showed, the mean value of SBP, DBP, FBS, TSB, LDL, were significantly higher and levels of albumin, and HDL were significantly lower in GSD group compared with healthy group. While other parameters not changed significantly.

CONFLICT OF INTERESTS

None.

AUTHOR'S CONTRIBUTIONS

Data and sample collection, laboratory works, data analysis and draft writing were done by Seerwan Assi Raheem; Ismail Salih Kakey did supervision and review and editing.

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