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Evaluation of Hormonal and Biochemical Profiles in Obesity, Exercise, and Gastric Sleeve Groups

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ABSTRACT

Objective: *This study aimed to investigate the biochemical and metabolic alterations linked to exercise, obesity, and sleeve gastrectomy,* ***Method:*** *this study was conducted according using an analytical crosssectional design. The study included 90 adult males participated aged between 35-45 years. Participants were divided into three groups: the first group consisted of healthy athletes (n = 30), the second group consisted of obese individuals before bariatric surgery (n = 30), and the third group consisted of the participants who had undergone bariatric surgery and were assessed at a single time point 8-months post-surgery. The study period is from October 2024 to December 2025. All participants fasted for 8 to 12 hours before blood sampling. The laboratory tests included hormone metabolism, liver enzymes, renal function indicators, total oxidative stress markers, and complete blood count (CBC).* ***Results:*** *In comparison to the athlete and obese groups, the sleeve gastrectomy group showed a statistically significant alteration in CBC values, total oxidative stress levels, liver enzyme activity, renal function markers, and metabolic hormone concentrations at ($P \leq 0.05$). These results imply that after sleeve gastrectomy, hematological, biochemical, and hormonal markers are significantly altered.* ***Novelty:*** *The novelty of this study lies in the comprehensive comparison of hematological, biochemical, oxidative stress, hormonal profile, and inflammatory cytokines across the three groups.*

Bariatric surgery markedly improves oxidative stress, inflammatory status, metabolic biomarkers, and hormonal regulation compared with obesity, while regular exercise represents the most physiologically balanced metabolic state.

INTRODUCTION

Globally, obesity affects over one billion individuals worldwide suffer from obesity that involved more than just being overweight [1]. A complex global health issue, obesity is defined by characterized by abnormal fat accumulation that upsets endocrine and biochemical balance. Such dysregulation is not only a result but a continuing contributor to this cycle [2], [3]. A Few of the many comorbidities that are further exacerbated by such dysregulation are type 2 diabetes, heart and vascular disorders and numerous hormonal abnormalities. The World Health Organization's alarming statistics from highlight how urgent it is to understand and effectively address this widespread health issue.

Hormones that regulate hunger, metabolism, energy expenditure, and fat distribution combine intricately to cause obesity [4]. Energy balance is maintained by many hormones, such as leptin (the satiety hormone), ghrelin (the hunger hormone), insulin, and several adipokines secreted by fat cells, perform essential functions in maintaining energy balance [5]. This strictly regulated mechanism is frequently upset in obese people. For instance, higher leptin presence in blood is seen but the body may become resistant to its signals, resulting in constant hunger [6]. Similarly, insulin resistance another well-known feature of type 2 diabetes is common in the context of obesity in which cells became less responsive to insulin and plasma glucose concentration increases [7]. Furthermore, reproduction hormones levels are frequently changing in obese individuals, influencing fertility status and overall metabolic profiles. Currently, one of the best treatments for severe obesity is bariatric surgery, especially sleeve gastrectomy procedure. Beyond its effect on weight loss, it causes pronounced modifications in hormonal and biochemical markers, such as marked decreases in leptin levels and changes in ghrelin secretion as a result of partial stomach resection [8]. These modifications highlight the systemic advantages of surgical intervention by improving endocrine and metabolic profiles. Limited investigations evaluate simultaneously the hormonal and biochemical profiles of these three different groups, despite the substantial research on obesity, exercise, and bariatric surgery separately [9], [10], [11]. By evaluating and differentiating the metabolic and endocrine variations among obese people, physically active individuals, and gastric sleeve patients, this study fills this gap and highlights the varying outcomes of lifestyle modification and surgical intervention. It is anticipated that the results will enhancing understanding of the mechanisms underlying metabolic improvements and guide medical management plans for adiposity.

Aim of the Study

The aim of this study is to evaluate and compare hormonal (leptin and ghrelin), hematological and biochemical (liver and kidney function) profiles among obese individuals, physically active participants, and patients who have undergone gastric sleeve surgery.

METHODS AND MATERIALS

This study was conducted according using an analytical cross-sectional design, and included a total of 90 adult male participated aged between 35-45 years. Participants were divided into three groups: the first group consisted of healthy athletes (n = 30), the second group consisted of obese individuals before bariatric surgery (n = 30), and the third group consisted of the participants who had undergone bariatric surgery and were assessed at a single time point 8-months post-surgery. All participants fasted for 8 to 12 hours before blood sampling. Samples were collected during the specified study period after meeting the inclusion and exclusion criteria, smokers or alcohol consumption, those taking medications that may affect metabolic or inflammatory. Data sample collection began on November 19, 2024. Statistical analysis was performed using SPSS software. Data expressed as mean ± SD. One-way ANOVA was used to compare differences among

groups, followed by Tukey's post hoc test. A p-value of ≤0.05 was considered statistically significant.

Ethical Approval and Participants' Consent

This study was conducted in accordance with the ethical principles outlined in the Declaration of Helsinki for research involving human subjects. The study protocol was reviewed and approved by the Institutional Review Board (IRB) / Ethical Committee of the relevant university/institution in Iraq. All participants (including obese individuals, post-bariatric surgery patients, and athletes) were fully informed about the objectives and procedures of the study prior to enrollment. Written informed consent was obtained from each participant. Confidentiality and anonymity were strictly maintained, and participants were informed of their right to withdraw from the study at any stage without any consequences.

Sample Collection and Laboratory Investigations

Venous blood samples were collected from all participants after overnight fasting. Serum was separated using a centrifuge and stored at appropriate temperatures until laboratory analyses were performed.

The laboratory investigations included the following:

- CBC, Hormones: (Leptin and Ghrelin)
- Interleukins (Il-17 and Il-34)
- Liver enzymes (AST, ALT)
- Kidney Function Tests (Urea, Creatinine)

All laboratory analyses were performed using standardized and validated laboratory techniques in accordance with the manufacturer's instructions.

RESULTS AND DISCUSSION

Table 1. Comparison of BMI among EX, Obese, and Bariatric Surgery groups, different letters indicate statistically significant differences between groups (P-Value ≤ 0.05).

Parameter	Mean±SD			P-Value
	EX(G1)	Obese(G2)	Bariatric Surgery(G3)	
BMI(Kg/m2)	25.454 ± 4.193 [a]	39.826 ± 5.580 [b]	32.947 ± 6.070 [C]	G1 VS G2 0.000 G1 VS G3 0.000 G2 VS G3 0.000

The findings represented in Table (1) showed that the Bariatric Surgery group (G3) showed a pronounced decline in BMI when compared with the Obese group (G2), while G3 significantly increased when compared with G1. Also, G2 showed a significant increase in comparison to G1, as shown in Fig (1), reflecting differences in adiposity, energy expenditure, and metabolic efficiency among the groups.

While preoperative interventions in the Bariatric Surgery group may partially reduce BMI without reaching the levels observed in habitually active subjects, the observed are consistent with established evidence that sedentary or obese individuals

present elevated BMI compared with physically active counterparts. Surgical-induced fat mass loss, particularly visceral adipose tissue, optimized metabolic function, better glucose response, changes in diet and activity after surgery are consistent with the significant decrease in BMI in the Bariatric Surgery group.

These findings may reflect differences in marked weight decrease and the bariatric surgery exhibited significant weight loss. These results agree with studies reporting significantly higher BMI in obese participants relative to physically active controls and a partial decrease after bariatric procedures [12–15].

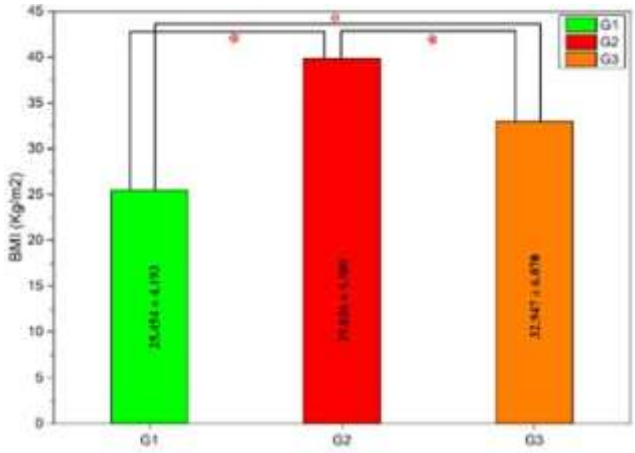

Figure 1. BMI Comparison of Obese, Bariatric Surgery, and Exercise Groups. When p-value ≤ 0.05 the stars refer to significance.

Table 2. CBC parameters Comparison in EX, Obese, and Bariatric Surgery groups, different letters indicate statistically significant differences between groups (P-Value ≤ 0.05).

Parameter	Mean±SD			P-Value
	EX(G1)	Obese(G2)	Bariatric Surgery(G3)	
WBC(103/µL)	a 7.013 ± 0.391	b 21.836 ± 4.173	c 10.760 ± 4.040	G1 VS G2 0.000 G1 VS G3 0.000 G2 VS G3 0.000
RBC(106/µL)	a 4.950 ± 0.363	b 5.200 ± 0.094	a 4.890 ± 0.355	G1 VS G2 0.002 G1 VS G3 0.439 G2 VS G3 0.000
HGB(g/dl)	a 14.343 ± 1.323	b 15.566 ± 0.442	a 14.050 ± 1.267	G1 VS G2 0.000 G1 VS G3 0.299 G2 VS G3 0.000

				G1 VS G2 0.000
HCT %	a 41.693 ± 8.244	b 48.333 ± 2.397	a 42.366 ± 4.230	G1 VS G3 0.638
				G2 VS G3 0.000
				G1 VS G2 0.000
MCV (fL)	a 88.466 ± 2.991	b 91.066 ± 1.855	a 88.900 ± 1.626	G1 VS G3 0.455
				G2 VS G3 0.000
				G1 VS G2 0.000
Neut%(%)	a 57.533 ± 1.716	b 86.133 ± 6.715	c 69.260 ±1 0.985	G1 VS G3 0.000
				G2 VS G3 0.000
				G1 VS G2 0.000
LYM %(%)	a 28.766 ± 0.897	b 11.713 ± 2.006	c 22.966 ± 9.506	G1 VS G3 0.000
				G2 VS G3 0.000
				G1 VS G2 0.299
PLT (103/µL)	a 298.300±49.763	a 288.500±36.839	b 339 ± 11.095	G1 VS G3 0.000
				G2 VS G3 0.000
				G1 VS G2 0.000
NLR	a 2.0002±0.0187	b 7.488 ± 0.891	c 3.910 ± 2.349	G1 VS G3 0.000
				G2 VS G3 0.000

The findings documented in Table (2) revealed a notable variation in (WBC, Neut%, NLR) levels among the study groups. As shown in Fig (2) the Obese group (G2) exhibited a substantial increase in comparison with EX (G1) and Bariatric Surgery ($p \leq 0.05$), This rise may be associated with the combined impact of immune system disturbance, prolonged adiposity and inflammation. The Obese group elevated WBC and neutrophil counts point to both the presence of chronic low-grade inflammation, characteristic feature of excessive e adiposity, and the stimulation of the innate immune response. The growth of adipose tissue increases the release of pro-inflammatory cytokines and chemokines, which active hemopoietic function activity and raise numbers of leukocytes in circulating, particularly neutrophils.

This neutrophilia is a sign of an environment that is pro-inflammatory may reflect to increased fat cells, immune cell accumulation, and metabolic status.

A significant decrease in WBC, neutrophils, and NLR were observed Following bariatric surgery, these improvements are probably linked to a substantial weight loss, reduced adipose tissue derived inflammatory signaling, and recovery of immune-metabolic balance which indicating attenuation of obesity-induced inflammatory stress. These results assessment with [16–19] [20–26].

The Exercise group consistently presented the lowest levels of WBC, neutrophils, and NLR, indicating a beneficial inflammatory status. Regular physical activity suppresses chronic inflammation through decreasing visceral fat mass, optimized insulin sensitivity, and increased release of antiinflammatory mediators.

Overall, the parallel changes observed in WBC, neutrophils, and NLR confirm that CBC-derived indices are not merely hematological markers but sensitive indicators of systemic inflammation and immune status, their improvement following bariatric surgery. confirms that interventions linked to body mass loss improve inflammatory control and immune balance [27–31].

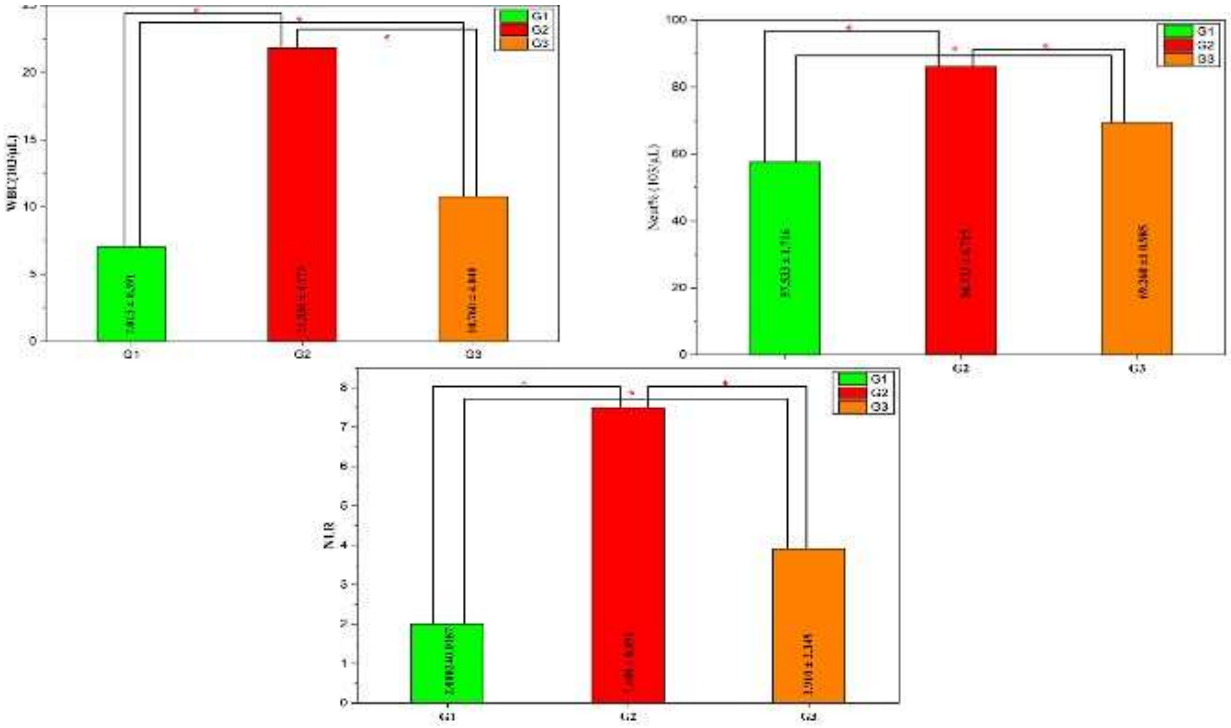

Figure 2. Comparison of (WBC, Neut%, NLR) among Obese, Bariatric Surgery, and Exercise groups.

The investigative findings in Table (2) signified (RBC, HGB, HCT, MCV) were modestly raised in Obese (G2) relative in comparison to EX ($p \leq 0.05$) and Bariatric Surgery G3, Bariatric Surgery showing equivalent values when compared with EX group($p \leq 0.05$), suggesting partial restoration post-surgery, as shown in Fig (3), these results may linked to raised RBC in obesity which may be correlated with greater oxygen requirement associated with increased body weight, while postoperative adjustment suggests restoration of balance of RBC production, this result assessment with [32–39]. Instead of

isolated variation in separate parameters, this general elevation is a coordinated hematological response to metabolic and inflammatory stress linked to obesity.

Change in erythrocyte may reflect metabolic and inflammatory adaptations associated with obesity. Sustained low intensity inflammation modulates bone marrow function through cytokine signaling, strengthening activation of red cell production and contributing to higher red cell indices, this adaptive response shows up as an increased RBC count, accompanied by elevated hemoglobin content in blood and hematocrit. This result assessment with [40–45].

By comparison, following considerable adiposity reduction, the surgically treated cohort exhibited marked attenuation of RBC, HGB, HCT, and MCV values, indicating attenuation of whole-body inflammatory burden, restored tissue oxygen supply, and re-established iron homeostasis. These changes indicate restoration of enhanced erythrocyte synthesis and mitigation of excess weight-induced erythrocytic strain associated with obesity. In a comparable manner, exercise-engaged participants demonstrated nadir measurements and most biological stable measurements, consistent with regular physical activity related improvements respiratory metabolic function, augmented circulatory performance, and reduced immune activation, this result assessment with [39,46–53] [54–57].

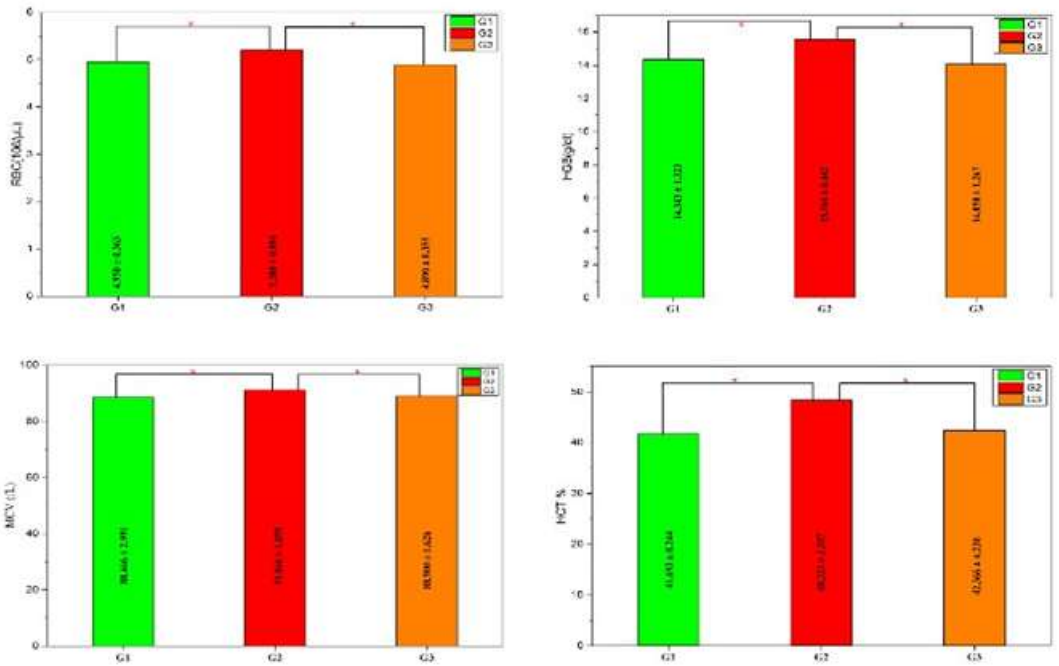

Figure 3. Comparison of RBC count, HGB, HCT, and MCV, among Obese, Bariatric Surgery, and Exercise groups.

Platelets (PLT, $10^3/\mu L$):

The Bariatric Surgery group (G3) had a significantly higher value than both the EX-group (G1) and the Obese group (G2), while the Obese were not significantly different when compared with EX group at ($p \leq 0.05$), as shown in Fig (4).This may be associated with obesity surgery stimulating the release of inflammatory cytokines, particularly interleukin-6 and TNF-α, which increases thrombopoietin, exhibited bone marrow activation and elevated PLT levels to support wound healing. Obesity may reflect

insufficient inflammation to significantly raise PLT, and exercise improves inflammation without a clear reduction in PLT, therefore no significant difference is shown between the two groups, this result agrees with [58–60], [61].

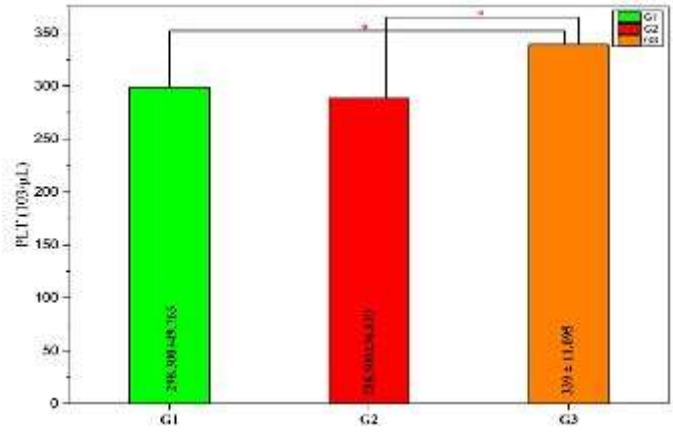

Figure 4. Comparison of PLT among Obese, Bariatric Surgery, and Exercise groups.

LYM %(103/µL):

The tabulated finding in Table (2) indicated (LYM) was prominently raised in Bariatric Surgery G3 and EX group G1 when compared with the obese group ($p \leq 0.05$),as illustrated visually in Fig (5), Obesity may linked to dysfunction and activation of the immune system as a result of chronic inflammation in the adipose tissue, where cytokines are secreted that lead to immune stress, disruption of lymphocyte proliferation, and an increase in their programmed death, which is reflected in a decrease in their number in the blood. In contrast, exercise improves cell mediated immunity by reducing inflammation, promoting blood flow and lymphocyte proliferation, and enhancing the effectiveness of the immune response. In bariatric surgery, the immune system shows recovery after rapid weight loss and reduces chronic inflammation, this result assessment with [62– 68].

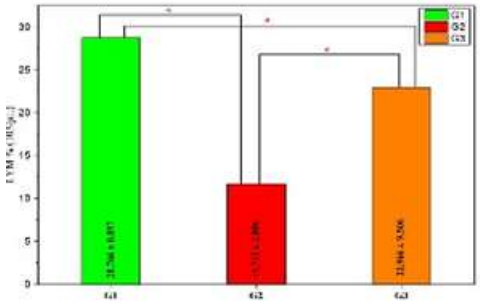

Figure 5. Comparison of LYM among Obese, Bariatric Surgery, and Exercise groups.

Table 3. Comparison of TAOC, TOS in EX, Obese, and Bariatric Surgery groups, different letters indicate statistically significant differences between groups (P-Value ≤ 0.05).

Parameter	Mean±SD			P-Value
	EX(G1)	Obese(G2)	Bariatric Surgery(G3)	
T-AOC(µmol/ml)	0.178 ± 0.059 a	1.086 ± 0.262 b	0.151 ± 0.201 a	G1 VS G2 0.000 G1 VS G3 0.584

The observations of T-AOC in Table (3) uncovered a significant reduction in both G3,G1 when evaluated against with G2 , at (p-value ≤0.05) , while a significant elevation TOS level in G2 when compared with G1,G3 , as shown in Fig (6), this results attributed to elevated TOS in obesity reflects a state of increased oxidative imbalance driven by excessive fat deposition, chronic low-grade inflammation, impaired mitochondrial activity, and amplified formation of reactive oxygen species (ROS). In obese individuals, adipocytes and invasive immune cells are known to contribute substantially to systemic oxidative burden, thereby elevating oxidant load.

Interestingly, the concurrent increase of T-AOC in the Obese group most likely reflects a physiological compensation to persistent oxidative stress, whereby endogenous antioxidant defenses are elevated in an attempt to counterbalance excessive ROS production.

Bariatric surgery is associated with a greater reduction in oxidative stress compared to physical activity, with a more pronounced antioxidant effect. It induces temporary stress that supports the body's defense systems, resulting in levels lower than those of obesity and higher than those of surgery, with less need for antioxidant boosting.

These findings highlight that whereas bariatric surgery leads to a considerable restoration of redox equilibrium through effective reduction of oxidant load, obesity is characterized by a dual state of heightened oxidative stress and compensatory antioxidant activation. Despite its advantages, Physical activity produces a distinct oxidative antioxidative profile reflecting physiological adaptation rather than pathological stress, these results assessment with [69, 69–72].

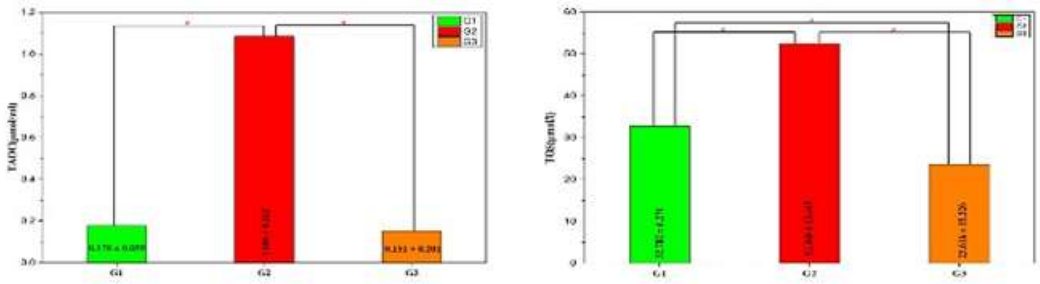

Figure 6. T-AOC, TOS Comparison of Obese, Bariatric Surgery, and Exercise Groups.

Table 4. AST, ALT levels Comparison in EX, Obese, and Bariatric Surgery groups, different letters indicate statistically significant differences between groups (P-Value ≤ 0.05).

Parameter	Mean±SD			P-Value
	EX(G1)	Obese(G2)	Bariatric Surgery(G3)	
AST(U/L)	a 32.922 ±30.094	b 57.716 ± 12.317	b 48.978 ± 15.694	G1 VS G2 0.000 G1 VS G3 0.004 G2 VS G3 0.108
ALT(U/L)	a 42.773±16.974	b 70.276 ± 14.782	a 45.438 ± 14.019	G1 VS G2 0.000 G1 VS G3 0.502 G2 VS G3 0.000

The findings outlined in Table (4) confirmed a measurable increase in AST enzyme activity in G3 when compared with G1, while absence of statistical variation between the G2 and G3 at ($p \leq 0.05$), as indicated in Fig (7),this result could be linked to the elevated AST in the Obese group reflects hepatic overload or minor liver injury associated with obesity, likely linked to hepatic steatosis and chronic metabolic strain, AST a non-hepatic enzyme affected by multiple factors.

The decrease in group G1 is attributed to routine physical activity. There is a general enhancement in hepatic and muscular metabolism and a reduction in cellular stress in relation to groups G2 and G3. Notwithstanding the improvement in liver function after surgery, the body remains under the influence of operative stress and metabolic rebalancing during this period. Furthermore, AST is impacted by non-hepatic sources such as muscle tissue.

Despite improvements in weight and systemic metabolism, the Bariatric Surgery group maintains elevated AST compared to physically active individuals, which may deflect early postsurgical metabolic restructuring or residual hepatic adjustment. The impact of obesity on liver function is evident and suggest that surgical management initiates metabolic improvements, though AST normalization may require longer-term follow-up. There was no detect a statistically significant difference between these two groups (G2, G3). This variation may be attributed to inconsistency in the postoperative follow-up interval, interindividual differences in hepatic recovery, and heterogeneous adherence to nutritional protocols. Such factors can transiently mask the expected improvement in transaminase levels, this result assessment with [73–75].

Based on the data in Table (4) a significantly decrease in ALT enzyme activity in G3 when against to G2 at ($p \leq 0.05$) but absence of significant variation was observed between the G1and G3 at ($p \leq 0.05$), as shown in Fig (7), ALT is a hepatic enzyme liberated into the bloodstream when liver cells are damaged or inflamed. It is elevated in obese individuals

associated with fat accumulation in the liver. During bariatric surgery, decrease in body mass and reduction of visceral fat decrease fat accumulation in the liver and reduce chronic inflammation. Additionally, enhanced insulin sensitivity reduces metabolic strain on the liver and lowers ALT activity. the ALT levels in the Bariatric Surgery group, being comparable to the physically active EX-group, suggest partial recovery of hepatic enzyme activity following bariatric treatment, reflecting improvement in liver function and metabolic stability postoperatively, this is what the results indicated.

Previous studies showing elevated ALT in obese individuals and significant reductions after bariatric procedures, indicating restoration of hepatic metabolic function, this result assessment with [76–79] .

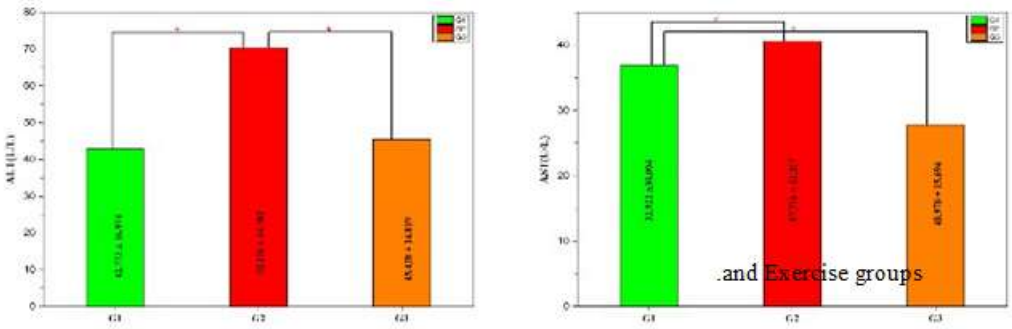

Figure 7. Comparison of AST, ALT among Obese, Bariatric Surgery,

Table 5. Comparison of Creatinine, Urea levels in EX, Obese, and Bariatric Surgery groups, different letters indicate statistically significant differences between groups (P-Value ≤ 0.05).

Parameter	Mean±SD			P-Value
	EX(G1)	Obese(G2)	Bariatric Surgery(G3)	
Creatinine (mg/dl)	1.075±0.318 a	2.123 ± 0.723 b	1.119 ± 0.429 a	G1 VS G2 0.000 G1 VS G3 0.743 G2 VS G3 0.000
Urea(mg/dl)	48.846±41.212 a	30.253± 9.387 b	21.858 ± 9.036 b	G1 VS G2 0.005 G1 VS G3 0.000 G2 VS G3 0.196

The results reported in Table (5) showed an exhibited markedly higher serum creatinine levels of (G2) Obese group when contrasted with both the EX-group (G1) and the Bariatric Surgery group (G3), whereas comparable values was observed between the EX and Bariatric Surgery groups at ($p \leq 0.05$), as presented in Fig (8), considerable body weight reduction after surgery typically leads to a decline in muscle mass, which decreases creatinine production independently of renal function changes since creatinine

is generated from skeletal muscle metabolism and this showed reduction in serum creatinine.

In early cases of obesity, elevated filtration rate appears, followed by glomerular stress and gradual renal injury over time, as the visceral fat accumulation linked to mechanical and metabolic stress on the kidneys, and increased insulin resistance leads to a decrease in eGFR and an increase in serum creatinine. Obesity also stimulates inflammatory cytokines that disrupt glomerular cell function, thus increasing permeability and renal damage, may reflect creatinine buildup in the blood. Bariatric surgery results in weight reduction, thereby reducing systemic inflammation, which in turn lower creatinine levels may reflect change in muscle mass and metabolic status; renal function cannot be fully assessed without eGFR or direct filtration markers. Exercise enhance glomerular perfusion, suppresses inflammation and oxidative damage, and improves blood pressure. Although athletes have higher muscle mass, their kidney function is often higher, resulting in lower or normal creatinine levels compared to obese individuals. This result assessment with [80–83].

furthermore, the results of serum urea levels showed that the EX-group exhibited the highest urea concentration, which was significantly higher than both the Obese group (G2) and the Bariatric Surgery group (G3). No significant difference was observed between the Obese and Bariatric Surgery groups at ($p \leq 0.05$), as shown in Fig (8), This result may reflect to the higher urea levels in the EX-group may reflect higher protein catabolism associated with increased physical activity, as active individuals often have enhanced protein metabolic activity and nitrogen metabolism, athletes often rely on a high-protein diet to support muscle building. Increased protein intake leads to increased urea production in the blood. In contrast, the lower urea levels in both the Obese and Bariatric Surgery groups may be attributed to reduced protein intake or altered nitrogen metabolism, which is commonly observed in obesity and post-bariatric surgery patients.

Additionally, in the Bariatric Surgery group, the reduced urea may reflect metabolic adaptations following surgical weight loss, including decreased hepatic urea synthesis linked to lower protein intake and altered amino acid catabolism, this result assessment with [84–86].

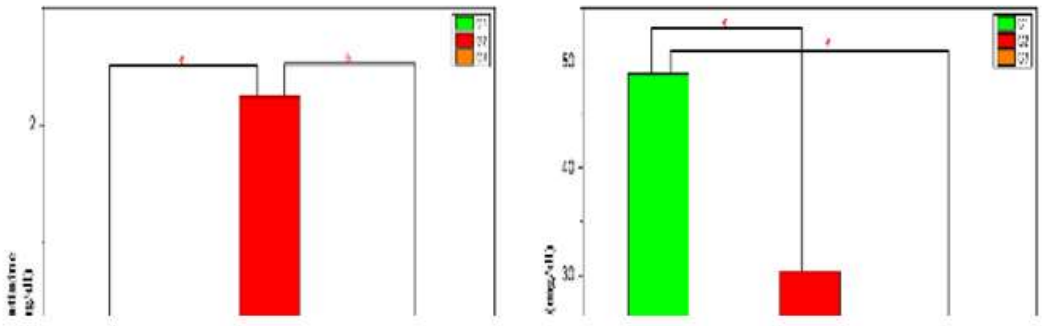

Figure 8. Creatinine, Urea Comparison of Obese, Bariatric Surgery, and Exercise groups.

Table 6. Comparing the levels of Ghrelin, Leptin in EX, Obese, and Bariatric Surgery groups, different letters indicate statistically significant differences between groups (P-Value ≤ 0.05).

Parameter	Mean±SD			P-Value
	EX(G1)	Obese(G2)	Bariatric Surgery(G3)	
Ghrelin (pg/ml)	589.066±77.207 a	384.75±47.694 b	149.083±49.530 c	G1 VS G2 0.000 G1 VS G3 0.000 G2 VS G3 0.000
Leptin (ng/ml)	9.112±3.469 a	45.564±15.226 b	24.415±11.602 c	G1 VS G2 0.000 G1 VS G3 0.000 G2 VS G3 0.000

The results reported in Table (6) indicated that Ghrelin have a notable decrease in G3 relative to both G2, G1, also showed a marked decrease in G2 versus G1 at ($p \leq 0.05$) , as depicted in Fig (9). Surgical excision of the gastric fundus, the primary location of ghrelin production, may be the cause of suppression contributes to reduced appetite, low caloric intake, and sustained postoperative weight loss, reinforcing ghrelin's critical role in the effectiveness of bariatric procedures. while reduced ghrelin in obesity reflects impaired hormonal regulation associated with chronic energy surplus. physically active have higher levels of ghrelin reflects a metabolic adaptation to elevated energy expenditure, to maintain energy balance and prevent excessive energy deficit ghrelin acts as an orexigenic compensatory signal.

In contrast, compared to the Exercise group, the Obese group had noticeably significantly lower ghrelin levels than the obesity is associated with impaired ghrelin regulation and blunted appetite signaling.

Conversely, as shown in Fig (9), leptin levels showed a significant elevation in G2 when comparison with both G1, G3, also G3 showed a significant increase versus G1 at ($p \leq 0.05$). The Bariatric Surgery group had a moderate level, the highest levels detected in the Obese group, and reduced levels in the Exercise group. Since leptin secretion is closely related to fat stores, the significantly elevated leptin in obesity reflects increased adipose tissue mass. Nevertheless, obese individuals often exhibit impaired leptin signaling having high circulating leptin, showed reduce satiety signaling and dysregulated energy expenditure. The Bariatric Surgery group reduced leptin levels show significant in the adipose tissue loss following surgical weight reduction, alongside partial restoration of leptin sensitivity, both of which improve metabolic regulation. With lower fat mass, preserved leptin sensitivity, and efficient energy homeostasis in physically active individuals, the Exercise group demonstrated the lowest leptin concentrations.

Leptin represents adipose-derived satiety signaling, whereas ghrelin primarily reflects gastricdriven appetite signaling. The significant divergence between these hormones between Exercise, Obese, and Bariatric Surgery groups illustrates that obesity and its management impact an interconnected endocrine system but rather than a single hormonal pathway, these finding assessment with [87–92], [93–96].

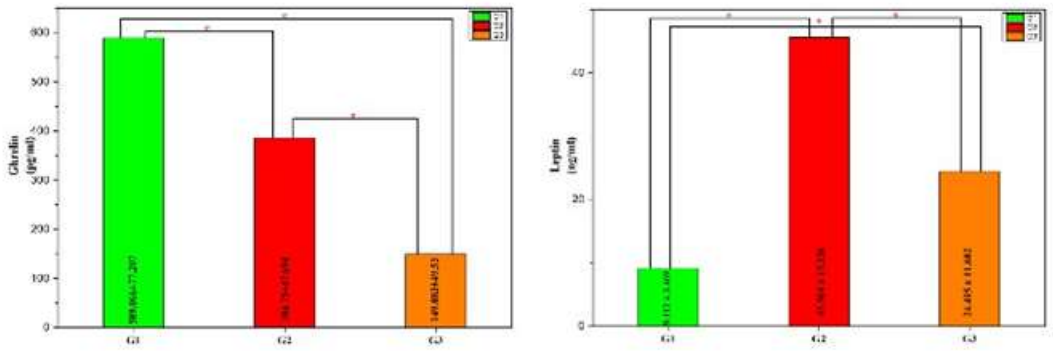

Figure 9. Ghrelin, leptin Comparison of Obese, Bariatric Surgery, and Exercise Groups.

Table 7. IL-17, IL-34 levels Comparison in EX, Obese, and Bariatric Surgery groups, distinct letters indicate statistically significant differences between groups (P-Value ≤ 0.05).

Parameter	Mean±SD			P-Value
	EX(G1)	Obese(G2)	Bariatric Surgery(G3)	
IL-17 (pg/ml)	17.935±12.014 a	466.238±162.877 b	96.441±96.105 c	G1 VS G2 0.000 G1 VS G3 0.007 G2 VS G3 0.000
IL-34 (pg/ml)	43.323±19.849 a	419.095±182.390 b	144.809±141.752 c	G1 VS G2 0.000 G1 VS G3 0.004 G2 VS G3 0.000

According to Fig (10), the current study showed that the levels of both IL-17 and IL-34 levels were statistically significant reduction in G3 relative to G2, whereas both IL-17 and IL-34 levels were significant elevation in G2 versus G1, which reflects clearly relationship between systemic inflammatory status adiposity, physical activity, and surgical weight loss.

Both of IL-17 and IL-34 concentration were greater in the Obese group, reflecting a pronounced inflammatory environment characteristic of obesity. IL-17 a key factor in the long-term inflammation, insulin resistance, and metabolic dysfunction that are frequently seen in obesity. As an active endocrine and immune organ increase fat tissue accumulation functions, promoting the activation of immune cells such as Th17 lymphocytes and macrophages, which contribute to elevated production of IL-17. Similarly, enhanced macrophage activation and inflammatory signaling persistence are elevated in IL-34 in obesity reflects, further reinforcing immune-metabolic imbalance.

The Exercise group exhibited the lowest IL-17 and IL-34 concentrations, consistent with the inflammation reducing effects of regular physical activity. Exercise is known to suppress proinflammatory cytokine production, enhance anti-inflammatory myokine release, and support immune balance.

The Bariatric Surgery group had intermediate levels of IL-17 and IL-34, significantly lower than in the Obese group but higher than the Exercise group, suggests gradual inflammatory resolution following bariatric surgery during postoperative recuperation. This reflects ongoing metabolic and immunological adaptation, weight loss contributing to reduced systemic inflammation, these result assessment with [97–99, 100].

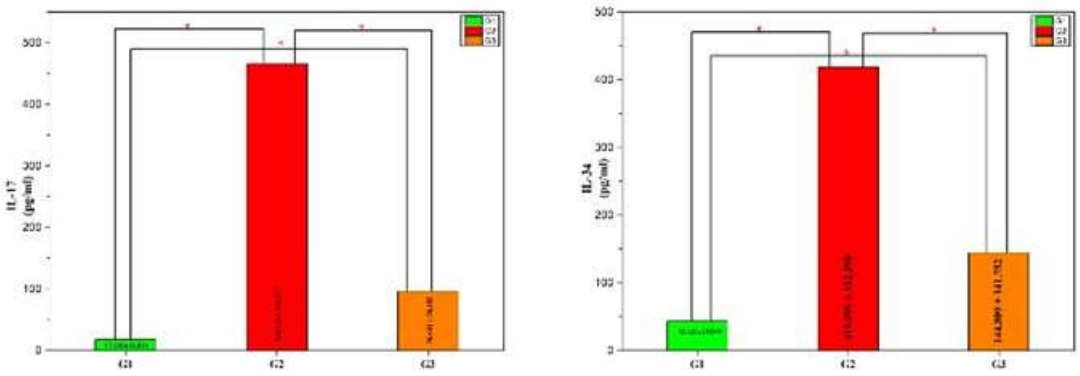

Figure 10. Comparison of IL-17, IL-34 among Obese, Bariatric Surgery, and Exercise groups.

CONCLUSION

Fundamental Finding: The present cross-sectional analysis delineates obesity as a state of profound metabolic dysregulation, characterized by increased oxidative stress, persistent low-grade inflammation, and multi-system perturbations encompassing hepatic and renal biomarkers, endocrine imbalance and altered hematological profiles. Regular exercise, is associated with a more regulated inflammatory and metabolic milieu, reinforcing its modulatory role in preventive function in preserving redox system and immunological homeostasis. Bariatric surgery was found to significantly alter in inflammatory, metabolic, and oxidative parameters. **Implication:** After bariatric surgery immunological and biochemical markers are essential for tracking metabolic recovery and informing postoperative management. Results demonstrate that bariatric surgery is an effective treatment metabolic disorders associated with obesity. These results show that regular exercise is essential and provide a physiological model that balances inflammation and metabolism. **Limitation:** Overall results reveal a gradual restoration of metabolic and immunological equilibrium after surgical reduction in weight, even though several indices, including AST and inflammatory mediators were marginally higher than in the homeostatic state during the postoperative phase. **Future Research:** Further studies are needed to clarify the persistence of elevated AST and inflammatory mediators during the postoperative phase and to determine the long-term trajectory of metabolic and immunological recovery after bariatric surgery.

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