Comparison of Liver Enzymes and Hormonal Profiles Between Cholecystectomized Patients and Non-Cholecystectomized Controls

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Abstract
Cholecystectomy is performed surgical procedure involving the removal of the gallbladder, that responsible for bile storage. The cholelithiasis, cholecystitis, and gallbladder cancer are gallbladder-related disorders that require cholecystectomy. The paper aims to compare liver enzymes levels and some gastrointestinal hormones between cholecystectomized Patients and controls. The study included 50 serum samples: 25 from Cholecystectomized patients and 25 from controls. The studied parameters include liver Enzymes (ALT, AST, ALP, and GGT). Also, the hormones (Cholecystokinin, Secretin, Gastrin, Insulin, Glucagon, and FGF19). The findings showed no significant differences between the patient and control groups for all liver enzymes at $P \leq 0.05$. Moreover, there are no significant differences in cholecystokinin, Secretin, Gastrin, Insulin, and Glucagon levels between the patient and the control. However, FGF19 decreased in the patient compared with the control. We can conclude that liver enzymes and some gastrointestinal hormones, except FGF19, don't show significant differences between Cholecystectomized Patients and Controls.

Keywords: Cholecystectomy, liver enzymes, cholecystokinin, Secretin, Gastrin, Insulin, Glucagon, and FGF19

Introduction

Cholecystectomy is a surgical procedure that involves the removal of the gallbladder, a small, pear-shaped organ located beneath the liver. The gallbladder plays a key role in storing and concentrating bile, which is essential for the digestion and absorption of dietary fats (**Liu et al., 2025).**

The procedure can be performed through a traditional open approach or, more commonly, via a minimally invasive laparoscopic approach. The laparoscopic method has become the standard due to its association with faster recovery, reduced postoperative pain, and shorter hospital stay (**Patil et al., 2024).** Cholecystectomy is used for patients suffering from gallbladder-related disorders. The most common condition is cholelithiasis, or gallstones, which can obstruct the bile ducts and cause severe abdominal pain, nausea, and digestive disturbances (**Jones et al. 2025).**

Other indications include cholecystitis (inflammation of the gallbladder) and gallbladder neoplasms. By removing the diseased organ, cholecystectomy alleviates symptoms, prevents biliary obstruction or pancreatitis, and improves the overall quality of life. Also, the procedure is considered prophylactic in high-risk patients who present with recurrent gallstone formation or anatomical abnormalities that predispose them to biliary complications (**Wang et al., 2021).**

The gallbladder is closely linked to liver function through its role in bile storage and regulated release into the duodenum. Following cholecystectomy, bile flow from the liver into the intestines can alter certain physiological processes. Studies have investigated whether this change influences liver enzyme profiles, particularly ALT, AST, ALP and GGT (**Jones et al., 2025).**

While most patients maintain normal liver function after surgery, transient elevations in these enzymes are sometimes observed postoperatively due to surgical stress, manipulation of hepatic tissues, or temporary alterations in bile flow (**Hosseinzadeh et al., 2025).** ALP and GGT, which are markers of biliary function, may be mildly elevated immediately after the procedure, reflecting adaptation to the continuous bile drainage. Over time, enzyme levels generally stabilise, although subtle differences persist between cholecystectomized patients and controls (**Gerussi, 2022).**

Moreover, cholecystectomy can alter the secretion of several gastrointestinal hormones, such as cholecystokinin and FGF19. The small intestine secretes CCK in response to dietary fats and proteins, thereby stimulating gallbladder contraction and pancreatic enzyme release (**Di Ciaula et al., 2018).** Following gallbladder removal, CCK levels may demonstrate a slightly elevated. Similarly, FGF19, a hormone involved in regulating bile acid synthesis and lipid metabolism, may show modest reductions because bile flow into the intestine is continuous rather than pulsatile. Also, many digestive and metabolic hormones, including gastrin, secretin, insulin, and glucagon, are generally unaffected in healthy individuals (**Barrera et al., 2015).**

Investigating the differences in liver enzyme levels and hormonal profiles between patients who have undergone cholecystectomy and those with intact gallbladders provides valuable insights into postoperative physiological adaptations. Such comparative analyses help to distinguish normal compensatory changes from pathological alterations that may indicate hepatic or metabolic complications. Furthermore, this comparison allows clinicians and

researchers to understand better the long-term effects of gallbladder removal on digestive efficiency, bile acid homeostasis, and metabolic regulation. By identifying the specific biomarkers most affected, healthcare professionals can optimise postoperative monitoring and implement targeted interventions to maintain metabolic health in cholecystectomized patients. Ultimately, this research contributes to a more comprehensive understanding of the systemic effects of cholecystectomy and informs both clinical practice and patient management strategies (**Saber et al., 2000).**

The study aims to compare the levels of liver enzymes, including ALT, AST, ALP, and GGT, between patients who have undergone cholecystectomy and individuals with intact gallbladders. To assess the impact of cholecystectomy on intestinal hormonal profiles, specifically focusing on cholecystokinin, FGF19, secretin, gastrin, insulin, and glucagon.

Materials And Methods

The study design:

The study included (50) serum samples that were collected from (50) individuals (adult males and females). (25) samples were collected from Cholecystectomized patients, and (25) samples were collected from the control group (healthy individuals). The studied parameters include liver Enzymes (ALT, AST, ALP, and GGT). Also, the hormones are included (Cholecystokinin, Secretin, Gastrin, Insulin, Glucagon, and FGF19.

Sample collection: Samples are collected in test tubes containing EDTA (an anticoagulant) and sent to the lab for the estimation of enzyme and hormone levels. At the lab, the samples are being prepared for plasma. The patient group consists of (11 adult females and 14 adult males) with an age ranged between (35-60), while the control group consist of (10 adult females and 15 adult males) with an age ranged between (35-60), as shown in table (1) .

Table (1): shows the number of the used males and females in the study

	Males	**Females**	**Total**
Patient group	**14**	**11**	25
Control group	**15**	**10**	25
Total	**29**	**21**	50

Liver Enzyme methods:

1. Alanine aminotransferase (ALT) IFCC kinetic UV (coupled LDH) method:

ALT catalyzes L-alanine + α-ketoglutarate → pyruvate + L-glutamate. Pyruvate is reduced to lactate by lactate dehydrogenase (LDH) using NADH; the rate of NADH oxidation is followed as a decrease in absorbance at 340 nm and is proportional to ALT activity (**Huang et al., 2006).**

Procedure (kinetic, 37 °C):

1. Collect venous blood into a plain tube; allow clotting, then centrifuge to obtain serum. Analyse within 4 hours at room temperature, or store at 2–8 °C for short-term storage; freeze at 20 °C for longer storage (**Flores, 2020).**
2. Bring reagents, samples, and cuvettes to 37 °C. Prewarm spectrophotometer/cobas/autoanalyser.
3. Pipette into cuvette: 1.0 mL reagent mix (buffer + substrate + NADH + LDH) + 10–50 µL serum. Mix and incubate 30–60 s to equilibrate.
4. Immediately record the decrease in absorbance at 340 nm every 15–30 s for 2–3 minutes (kinetic slope). Ensure linearity for at least two readings.
5. Calculate ΔA340/min (slope) and convert to activity using the NADH molar absorptivity (ε ≈ 6,220–6,317 $L \cdot mol^{-1} \cdot cm^{-1}$ at 340 nm, depending on reference temperature) and sample dilution (**McComb et al., 1976**).:

$$Activity(U/L) = \frac{\Delta A/\min \times V_{total}}{\varepsilon \times l \times V_{sample}} \times 10^6$$

2. Aspartate aminotransferase (AST) IFCC kinetic UV (coupled MDH) method: AST converts L-aspartate + α-ketoglutarate → oxaloacetate + L-glutamate. Oxaloacetate is reduced by malate dehydrogenase (MDH) using NADH; the rate of NADH oxidation (decrease at 340 nm) is proportional to AST activity. The assay format and calculation parallel ALT (**Huang et al., 2006).**

Procedure: Identical workflow to ALT: prewarm to 37 °C, mix reagent + serum, record ΔA340/min, convert to U/L using NADH ε and sample/cuvette geometry. Document assay lot, analyzer model, and traceable calibrator.

3. Alkaline phosphatase (ALP) p-nitrophenyl phosphate (pNPP) colorimetric assay: ALP hydrolyzes p-nitrophenyl phosphate (pNPP) to p-nitrophenol (pNP). Under alkaline conditions, pNP is yellow and is measured spectrophotometrically (commonly at 405 nm). Reaction rate is proportional to ALP activity. The pNPP method is widely used and adaptable to manual or automated analysers (**Kanta et al., 2021).**

Procedure:

1. Serum preparation then prewarm reagents to 37 °C (or 25 °C if using room-temperature reference).
2. Mix 1.0 mL reagent (pNPP buffer) with 10–50 µL serum, immediately measure the increase in absorbance at 405 nm at fixed intervals for 2–10 min and calculate ΔA405/min. For endpoint: incubate for a fixed time (e.g., 15–30 min), then add the stop solution and read at 405 nm.

3. Convert ΔA/min to U/L using the molar absorptivity of pNP at the chosen wavelength/temperature and account for dilutions and path length. Typical ALP reference materials and calibrators should be used to derive final units.

4. Gamma-glutamyl transferase (GGT) kinetic colourimetric method: GGT transfers the γ-glutamyl moiety from a donor (e.g., γ-glutamyl-p-nitroanilide) to an acceptor; the released p-nitroaniline (pNA) is chromogenic and measured at ≈405 nm. The kinetic rate of pNA formation is proportional to GGT activity. Classic methods (Persijn, Tamaoki, Fossati) and modern automated kinetic adaptations are standard (**Persijn et al., 1976).**

Procedure:

1. Prepare serum as above. Prewarm to assay temperature.
2. Mix the reagent with the sample, immediately measure the increase in absorbance at 405 nm at regular intervals for 2–5 minutes.
3. Determine ΔA405/min and convert to U/L using standard curves or molar absorptivity of pNA, adjusting for path length and dilution. Many automated analysers provide calibrated GGT output reports that include the analyser and assay versions.
5. General pre-analytical and analytical considerations (sample handling, calibration, units).

The laboratory techniques used for quantifying gastrointestinal Hormones

1. Cholecystokinin (CCK): CCK is quantified using a competitive ELISA or RIA utilizing high-affinity antibodies specific to biologically active CCK peptides (e.g., CCK-8, CCK-33). Signal intensity is inversely proportional to CCK concentration.

Procedure:

1. Collect blood in EDTA-treated aprotinin tubes; immediately centrifuge at 4 °C to prevent peptide degradation.
2. Use commercial competitive CCK ELISA kits. Add 50–100 µL plasma to wells pre-coated with CCK antibodies.
3. Incubate for 1–2 h at room temperature (or 4 °C overnight, depending on the kit).
4. Add enzyme-linked detection reagent; wash.
5. Add the chromogenic substrate (TMB), stop the reaction, and read absorbance at 450 nm.
6. Generate a standard curve using known CCK calibrators and calculate sample levels.

2. Secretin: Secretin is measured via competitive immunoassay using anti-secretin antibodies. Due to low plasma concentration, assays require high analytical sensitivity.

Procedure:

1. Draw fasting blood into chilled EDTA tubes containing protease inhibitors.
2. Centrifuge at 4 °C; freeze plasma at −80 °C.
3. Add the sample to antibody-coated wells, together with the secretin-HRP conjugate.
4. After incubation and washing, add substrate and measure absorbance at 450 nm.
5. Quantify concentration using a standard curve.

3. Gastrin: Gastrin is determined using competitive or sandwich immunoassays. Chemiluminescent detection increases sensitivity for low-level measurements.

Procedure:

1. Collect fasting serum; avoid hemolysis.
2. Add serum to wells coated with anti-gastrin monoclonal antibodies.
3. Add enzyme- or acridinium-labelled secondary antibody (CLIA).
4. Incubate, wash, and add chemiluminescent substrate.
5. Read the signal on the automated analyser; derive the concentration from the calibration curve.

4. Insulin: The immunoassay using monoclonal antibodies against different insulin epitopes. Signal intensity is directly proportional to insulin concentration.

Procedure:

1. Serum is collected after an 8-hour fast.
2. Add 10–50 µL serum to wells coated with insulin capture antibody.
3. Add enzyme-linked detection antibody (HRP or acridinium).
4. Incubate and wash.
5. Add substrate (TMB or chemiluminescent reagent).
6. Read absorbance/chemiluminescence and quantify using a standard curve.

5. Glucagon: Modern ELISA kits use monoclonal antibodies specific for glucagon (1-29), avoiding cross-reactivity with proglucagon fragments.

Procedure:

1. Use EDTA plasma containing aprotinin (very important due to peptide instability).
2. Add sample to wells pre-coated with glucagon capture antibody.
3. Add biotinylated detection antibody + streptavidin-HRP.
4. Wash, add TMB substrate, stop, and read absorbance at 450 nm.
5. Calculate using the standard curve.

6. Fibroblast Growth Factor 19 (FGF19): FGF19 is quantified using a two-site ELISA with capture and detection antibodies directed against distinct epitopes on the FGF19 protein.

Procedure:

1. Collect serum after overnight fasting; centrifuge and store at −80 °C.

2. Add 50–100 µL serum to wells coated with anti-FGF19 capture antibody.
3. Incubate 1–2 hours.
4. Add HRP-conjugated detection antibody.
5. After washing, add TMB substrate; stop reaction; read absorbance at 450 nm.
6. Use a 4-parameter logistic (4PL) standard curve for quantification.

Statistical analysis: The values in tables are expressed as mean ± standard error. The Statistical analysis is carried out by XLSTAT 2020.

Results

According to our results, mean levels ± standard error of Alanine transaminase (ALT), Aspartate transaminase (AST), Alkaline phosphatase (ALP), and Gamma glutamyl transferase (GGT) were (15.5 ± 3.6 IU/L), (17.3 ± 3.1 IU/L), (93.4 ± 6.5 IU/L) and (30.9 ± 5.3 IU/L) in the control group. In comparison, the mean levels of these enzymes were (16.3 ± 2.9 IU/L), (15.5 ± 3.2 IU/L), (89.8 ± 5.3 IU/L), and (28.1 ± 5.1 IU/L) in the patient group, at P≤0.05, as shown in Table (2).

Based on our findings, there are no significant differences between the patient and control groups for all liver enzymes (ALT, AST, ALP, and GGT) at P≤0.05, as shown in Table (2) and Figure (1).

Table (2): shows the mean levels of the liver enzymes in the study groups

liver enzymes	control group (IU/L)	patient group (IU/L)
Alanine transaminase (ALT)	**15.5 ± 3.6 A**	**16.3 ± 2.9 A**
Aspartate transaminase (AST)	**17.3 ± 3.1 B**	**15.5 ± 3.2 B**
Alkaline phosphatase (ALP)	**93.4 ± 6.5 C**	**89.8 ± 5.3 C**
Gamma-glutamyl transferase (GGT)	**30.9 ± 5.3 D**	**33.1 ± 5.1 D**

Similar letters indicate no significant difference, while different letters indicate significant differences.

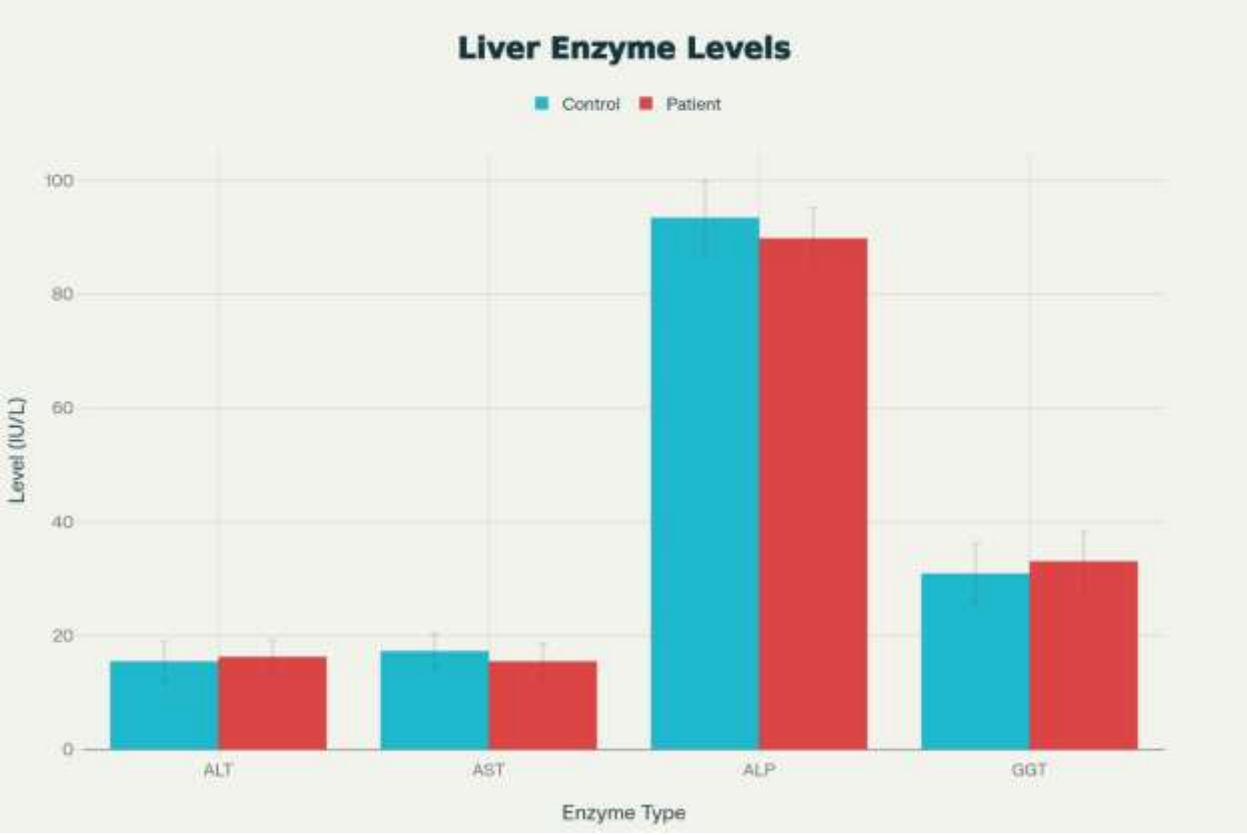

Figure (1): shows the columns represent the levels of the liver enzymes in the study groups.

According to our results, the mean level of Cholecystokinin, Secretin, Gastrin, Insulin, Glucagon and FGF19 in the control group was 36.3 ± 0.2 pg/mL, 35.6 ± 0.9 pg/mL, 64.5 ± 7.5 pg/mL, 9.3 ± 1.4 µIU/mL, 93.8 ± 6.6 pg/mL and 88.4 ± 7.1 pg/mL, respectively. The mean level of Cholecystokinin, Secretin, Gastrin, Insulin, Glucagon and Fibroblast Growth Factor 19 (FGF19) in the patient group were 38.9 ± 1.7 pg/mL, 33.9 ± 2.3 pg/mL, 66.5 ± 5.3 pg/mL, 9.5 ± 1.8 µIU/mL, 94.1 ± 4.6 pg/mL and 65.7 ± 5.3 pg/mL, respectively.

The findings indicate that there are no significant differences in the mean levels of cholecystokinin, Secretin, Gastrin, Insulin, and Glucagon between the control and patient groups. However, Fibroblast Growth Factor 19 (FGF19) decreased in the patient group compared with the control group (P≤0.05), as shown in Table (3) and Figure (2).

Table (3): shows the mean levels of the hormones in the study groups

The hormone	control group (pg/mL)	patient group (pg/mL)
Cholecystokinin (CCK)	**36.3 ± 0.2 pg/mL A**	**38.9 ± 1.7 pg/mL A**
Secretin	**35.6 ± 0.9 pg/mL B**	**33.9 ± 2.3 pg/mL B**
Gastrin	**64.5 ± 7.5 pg/mL C**	**66.5 ± 5.3 pg/mL C**
Insulin	**9.3 ± 1.4 µIU/mL D**	**9.5 ± 1.8 µIU/mL D**
Glucagon	**93.8 ± 6.6 pg/mL E**	**94.1 ± 4.6 pg/mL E**
Fibroblast Growth Factor 19 (FGF19)	**88.4 ± 7.1 pg/mL F**	**65.7 ± 5.3 pg/mL f**

Similar letters indicate no significant difference, while different letters indicate significant differences.

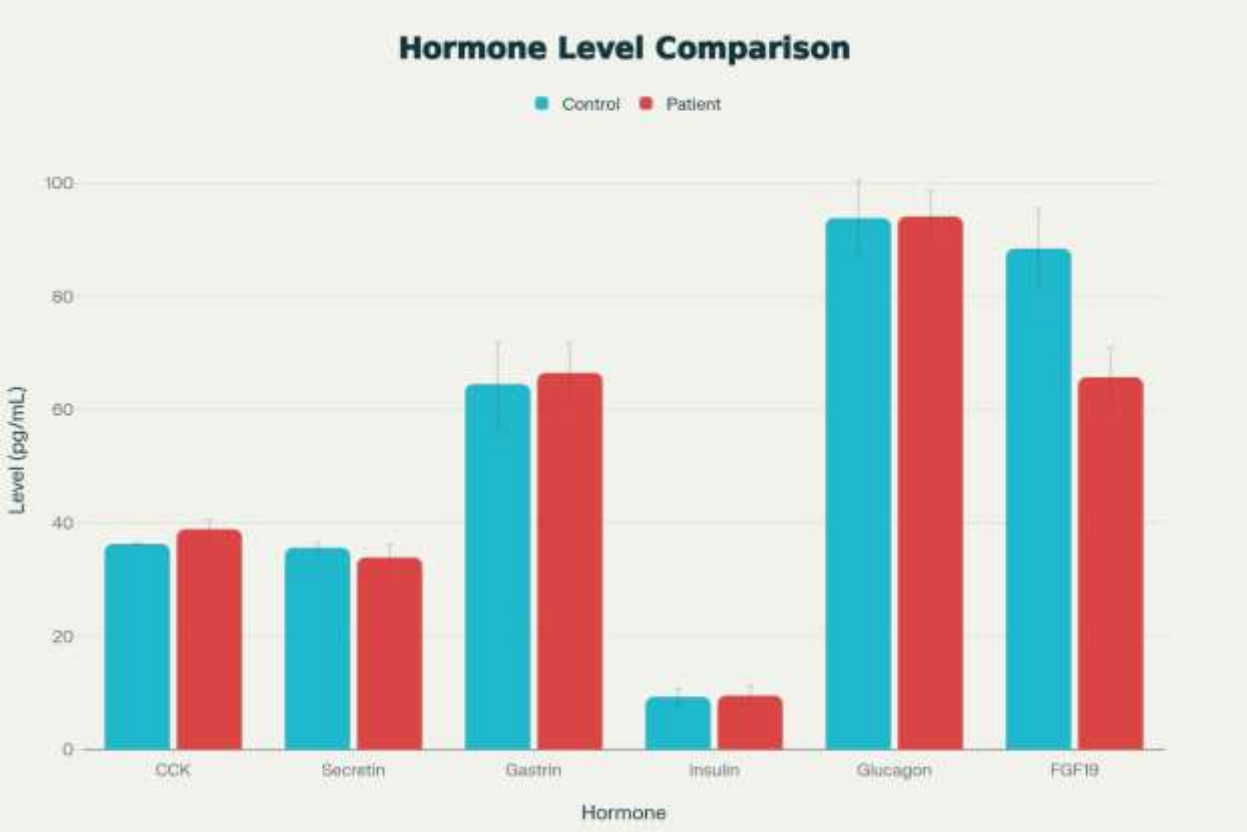

Figure (2): shows the columns represent the levels of some hormones in the study groups.

Discussion

Cholecystectomy is one of the most frequently performed abdominal operations worldwide, primarily indicated for symptomatic cholelithiasis and cholecystitis. Although the procedure effectively relieves biliary colic and prevents recurrent gallstone complications, it produces measurable changes in host physiology that extend beyond simply removing a bile reservoir (Salati SA., **2002).**

The absence of the gallbladder alters the timing and composition of bile delivery to the intestine, potentially increasing circulating secondary bile acids. Such bile-mediated changes have been linked experimentally and epidemiologically to metabolic consequences (**Liu et al., 2022).**

Based on our findings, there are no significant differences between the patient and control groups for all liver enzymes (ALT, AST, ALP, and GGT).

Multiple reports and studies indicate that an uncomplicated cholecystectomy does not cause persistent derangement of liver enzymes, which is consistent with our finding that ALT, AST, ALP, and GGT did not differ significantly between cholecystectomized patients and non-cholecystectomized controls. Several clinical series have shown that changes in liver function tests after laparoscopic cholecystectomy are typically transient, related to the perioperative period, and resolve within days to weeks without long-term biochemical sequelae in patients with previously normal liver function (**Giakoustidis et al., 2024).**

In another study measuring liver enzymes before and after laparoscopic cholecystectomy, postoperative rises in ALT and AST were consistently observed on days 1–2, with values returning to baseline by day 7–10. There are significant

early increases in aminotransferases that normalized within 48–120 hours, and emphasised that these disturbances were self-limited and clinically silent when the liver was otherwise healthy. Although two-thirds of patients had postoperative abnormalities in AST, ALT or GGT, repeat testing three weeks later showed complete normalisation, leading to conclude that routine post-cholecystectomy liver testing is unnecessary in asymptomatic individuals. These data support the concept that, once recovery is complete, the biochemical profile of cholecystectomized patients should be indistinguishable from that of non-operated controls, as observed in our study (**Saber et al., 2000).**

The mechanisms proposed for these transient elevations further explain why long-term differences are not expected. Experimental and clinical work implicates carbon dioxide pneumoperitoneum, reduced hepatic blood flow, traction on the liver, and manipulation of the biliary tree as causes of short-lived hepatocellular stress. When insufflation is discontinued, and circulation normalises, hepatocytes recover and enzyme release diminishes, leading to the restoration of baseline ALT and AST. ALP and GGT, which reflect cholestasis and biliary epithelial injury, may also rise briefly but show a similar pattern of normalization once oedema and transient obstruction resolve (**Ahmad NZ., 2011).**

Nevertheless, some reports could be interpreted as conflicting with our findings. Several series document significant postoperative enzyme elevations and highlight correlations with operative duration, higher pneumoperitoneum pressure, or co-existing liver disease. Meta-analytic and scoping reviews confirm that a large proportion of patients develop biochemical abnormalities in the early days after laparoscopic cholecystectomy, sometimes exceeding twice the upper reference limit. However, these studies almost uniformly describe the changes as transient and of "statistical but not clinical" significance, with normalization within 7–10 days in individuals without baseline hepatic dysfunction (**Singh et al., 2019).**

Another factor is patient selection. Many earlier investigations included individuals with symptomatic cholelithiasis, subclinical cholestasis or metabolic comorbidities that can influence enzyme levels even before surgery. By contrast, our control and cholecystectomized groups were matched and free of active hepatobiliary disease, minimizing confounding from underlying pathology. Moreover, liver enzymes are known to have considerable biological variability and limited sensitivity for mild chronic injury, which means that subtle metabolic effects of cholecystectomy, such as changes in bile acid circulation or risk of fatty liver, may occur without shifting ALT, AST, ALP or GGT outside the reference range (**Maleknia et al., 2020).**

There is strong evidence supporting our observation, with no significant long-term differences in standard liver enzyme levels between cholecystectomized patients and non-cholecystectomized controls. Early postoperative elevations are well documented but transient, largely attributable to pneumoperitoneum and surgical manipulation, and they resolve without lasting biochemical damage in patients with normal baseline liver function (**Salati SA., 2022).**

The findings indicate that there are no significant differences in the mean levels of cholecystokinin, Secretin, Gastrin, Insulin, and Glucagon between the control and patient groups. However, Fibroblast Growth Factor 19 (FGF19) was lower in the patient group than in the control group.

The absence of significant differences in the mean levels of cholecystokinin (CCK), secretin, gastrin, insulin and glucagon between cholecystectomized patients and non-cholecystectomized individuals is consistent with current understanding of the gut–liver axis. These hormones are primarily produced by intestinal and pancreatic cells in response to nutrient exposure, whereas the gallbladder functions mainly as a bile reservoir rather than an endocrine organ. Consequently, gallbladder removal alone is not expected to permanently alter their basal secretion patterns when the remaining biliary tract and intestine are intact. This explains why, in a stable postoperative setting, fasting hormone concentrations often overlap with those of healthy controls (**Yılmaz et al., 2022).**

Several physiological and clinical studies examining CCK after cholecystectomy have reported comparable fasting plasma levels between patients and controls, with differences mainly in postprandial responses. In some cohorts, CCK peaks after a meal are modestly higher or more prolonged in cholecystectomized patients, reflecting compensation for the loss of gallbladder storage and the need to coordinate continuous bile flow with intestinal motility. However, these dynamic changes do not necessarily shift overall mean concentrations when measured under standardised fasting conditions, as in your dataset. Similar reasoning applies to secretin and gastrin, whose release is driven by luminal pH and nutrient content, and to insulin and glucagon, which are tightly regulated by glucose homeostasis rather than by gallbladder status. Therefore, the lack of between-group differences in these classical gut and pancreatic hormones is biologically plausible (**McDonnell et al., 2002).**

In contrast, the observed decrease in fibroblast growth factor 19 (FGF19) in the cholecystectomized group aligns closely with more recent work on bile acid signalling. Human and experimental data show that FGF19 is produced not only by ileal enterocytes but also by the biliary epithelium, with the gallbladder expressing markedly higher levels than the distal small intestine and concentrating FGF19 in bile. After cholecystectomy, this important source is removed, and bile is delivered more continuously to the intestine, altering activation of the farnesoid X receptor (FXR) and the negative feedback loop that FGF19 exerts on hepatic bile acid synthesis. Clinical studies have demonstrated increased bile acid production rates and a blunted circadian peak of circulating FGF19 several months after gallbladder removal, supporting the concept that cholecystectomy selectively reduces systemic FGF19 while leaving other enteroendocrine hormones largely unchanged (**Farrugia et al., 2024).**

Reported discrepancies in the literature can often be traced to differences in study design and patient characteristics. Studies that document higher CCK or altered insulin and glucagon responses after cholecystectomy typically assess dynamic postprandial curves, include participants with obesity or insulin resistance, or focus on early postoperative

or symptomatic states, all of which can modulate hormone secretion. By contrast, cross-sectional comparisons of fasting, clinically stable individuals with and without prior cholecystectomy tend to show minimal differences in CCK, secretin, gastrin and pancreatic hormones, while still detecting altered bile acid FGF19 signalling. Taken together, current evidence supports a model in which cholecystectomy does not meaningfully disturb basal secretion of classical gut and pancreatic hormones but does lower FGF19 due to loss of gallbladder-derived production and re-patterning of enterohepatic bile acid cycling, in agreement with the pattern observed in your study (**Di Ciaula et al., 2018).**

Conclusion

Cholecystectomy is a surgical procedure that is done for many cases, such as cholelithiasis, cholecystitis, and gallbladder cancer, to remove the gallbladder to prevent complications. There are no significant differences between the patient and control groups for all liver enzymes. Moreover, there are no significant differences in cholecystokinin, Secretin, Gastrin, Insulin, and Glucagon levels between the patient and the control, except for FGF19, which showed a lower level in the patient compared with the control. We can conclude that liver enzymes and some gastrointestinal hormones, except FGF19, don't show significant differences between Cholecystectomized Patients and Controls. Overall, cholecystectomy appears to leave long-term liver enzymes and most gastrointestinal hormones unchanged, with only FGF19 showing a notable reduction. These findings suggest that gallbladder removal produces limited lasting biochemical effects, supporting the stability of hepatic and hormonal function after recovery.

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