

Changes in lipid, liver, and renal profiles among patients with severe covid-19 during and after hospitalization at Saint Peter Specialized Hospital, Addis Ababa

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Abstract

Background: COVID-19 progression affects multiple organ systems, and abnormalities in lipid, liver, and renal function tests have been reported. This study aimed to evaluate changes in organ function and lipid profiles among patients with severe COVID-19 during hospitalization and at discharge.

Methods: A follow-up study was conducted at St. Peter Specialized Hospital from January 1 to April 30, 2021, involving 162 hospitalized COVID-19 patients. Five milliliters of venous blood were collected at admission and again at discharge. Lipid, renal, and liver function tests were performed using the Cobas 311 analyzer. Data were entered and analyzed using SPSS version 25.

Results: Significant mean differences were observed in total cholesterol (20.13 mg/dL; 95% CI: 13.41–26.84; $P < 0.001$), HDL (7.53 mg/dL; 95% CI: 5.24–9.81; $P < 0.001$), and LDL (0.10 mg/dL; 95% CI: 0.06–0.14; $P < 0.001$) between admission and discharge. Albumin levels significantly increased at discharge, while ALT levels significantly decreased ($P < 0.05$).

Conclusion: Dyslipidemia and hypoalbuminemia were observed during the acute phase of severe COVID-19. These findings suggest that severe COVID-19 induces significant alterations in lipid metabolism and liver function. Monitoring and managing lipid levels may improve clinical outcomes and reduce complications. Integrating lipid management into COVID-19 treatment protocols could enhance patient recovery and care effectiveness.

Keywords: COVID-19, Lipid profile, Liver function test, Renal function test

Introduction

Coronavirus disease 2019 (COVID-19) is an infectious disease caused by the novel severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2). Since its emergence, the pandemic has affected millions of individuals across the globe. A substantial proportion of cases progress rapidly to acute respiratory distress syndrome (ARDS), multiorgan failure, and septic shock, often resulting in increased mortality (Sun et al. 2020; Khan et al. 2020).

Lipid metabolism plays a critical role in viral replication and immune response, making it a potential target for antiviral strategies. In particular, SARS-CoV-2 is thought to exploit host lipid metabolic pathways and cellular membrane components for its life cycle. High-density lipoprotein (HDL), for instance, may facilitate viral entry by interacting with the scavenger receptor class B type 1 (SR-B1) on host cells (Koçar et al. 2021). Although SARS-CoV-2 appears to affect lipid metabolism, data on alterations in HDL-C levels in COVID-19 patients remain limited. Several studies have reported that total cholesterol (TC) and low-density lipoprotein cholesterol (LDL-C) levels significantly decrease in infected individuals (Wang et al. 2020).

Hepatic involvement in COVID-19 may result from several mechanisms, including direct cytopathic effects of the virus, a dysregulated immune response, sepsis, or drug-induced liver injury. The liver, particularly

cholangiocytes, which express high levels of angiotensin-converting enzyme 2 (ACE2) receptors, is a known target of SARS-CoV-2. Additionally, the virus can exacerbate pre-existing liver conditions, potentially leading to hepatic decompensation or acute-on-chronic liver failure, which are associated with high mortality rates (Jothimani et al. 2020).

COVID-19 has also been linked to multiple complications, including myocardial injury, heart failure, acute kidney injury (AKI), and electrolyte imbalances. Risk factors for severe disease and mortality include advanced age, male sex, and pre-existing comorbidities such as cardiovascular disease, diabetes, chronic kidney disease, and chronic liver disease. These complications often correlate with disease severity and are reflected in abnormal laboratory parameters such as liver enzymes, renal function tests, cardiac markers, and lactate dehydrogenase (LDH) levels (Li et al. 2021; Zaim et al., 2020).

Studies from various regions support the association between COVID-19 and metabolic alterations. A descriptive study in Northwest Mexico found significant changes in lipid profiles among both critical and noncritical COVID-19 patients, including reduced levels of TC, LDL, and HDL, and elevated triglycerides (TG) (Osuna-Ramos et al. 2020). Similarly, a prospective cohort study involving 701 hospitalized COVID-19 patients in Wuhan, China, reported elevated serum

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creatinine in 14.4% and elevated blood urea nitrogen in 13.1% of patients (Cheng et al. 2020). Additional renal manifestations such as proteinuria, hematuria, and mild creatinine elevation have been linked to direct viral kidney involvement and systemic multiorgan failure (Benedetti et al. 2020).

Furthermore, a meta-analysis by Wu Y et al. (2020) reported that the pooled incidence of abnormal liver biochemical markers was 27.2% at admission and 36% during hospitalization. These abnormalities were significantly associated with disease severity and clinical outcomes.

Given the clinical implications of these findings, this study aimed to evaluate changes in common laboratory parameters—including lipid profiles, liver function tests, and renal function tests—at the time of admission and at discharge among patients with severe COVID-19 admitted to St. Peter Specialized Hospital in Addis Ababa, Ethiopia. The results are intended to contribute to improved risk stratification and inform clinical management strategies for COVID-19.

Materials and Methods

Study Area and Design

A prospective cohort study was conducted from January 1 to April 30, 2021, at St. Peter Specialized Hospital, located in Gulele Subcity, Addis Ababa, Ethiopia. The hospital, established in 1953, operates under the Ministry of Health of the Federal Democratic Republic of Ethiopia and serves as a referral center for COVID-19 patients.

Eligibility Criteria

All newly admitted patients with confirmed COVID-19 during the study period were considered for inclusion. Patients with known histories of chronic kidney disease, chronic liver disease, or dyslipidemia were excluded to minimize confounding factors.

Sample Size Determination and Sampling Method

The sample size was calculated using a two-sample means comparison formula, based on a previously reported difference in HDL levels (6.6 mg/dL) [Qin C et al. 2020]. With 80% power and a 99% confidence level, the minimum required sample size was estimated at 147. To maximize reliability, all 162 eligible patients admitted during the study period were included.

2.4. Sample Collection and Laboratory Analysis. Approximately 5 mL of venous blood was aseptically collected from each participant by trained laboratory technologists, in the morning following at least 8 hours of fasting. Samples were drawn into serum separator tubes or plain tubes, labeled with a unique participant ID. After allowing the blood to clot for 30 minutes at room temperature, the samples were centrifuged at 300 g for 5 minutes to separate the serum. Biochemical analyses were performed on the same day using a Cobas c311 fully automated clinical chemistry analyzer (Roche

Diagnostics, Indianapolis, USA), in accordance with the manufacturer's instructions.

- Lipid profile:
 - Total cholesterol (TC), HDL-C, LDL-C, and triglycerides (TG) were measured using enzymatic colorimetric methods (cholesterol oxidase, phenol-4-aminoantipyrine peroxidase, direct enzymatic methods, and glycerol phosphate oxidase peroxidase, respectively).
- Liver function tests:
 - Alanine aminotransferase (ALT), aspartate aminotransferase (AST), and albumin (ALB) levels were measured based on enzymatic activity using IFCC-recommended methods.
 - Total and direct bilirubin (BILT and BILD) were assessed using the diazo colorimetric method.
- Renal function tests:
 - Serum creatinine and urea were analyzed using enzymatic methods.

Data Quality Control

Patient preparation and sample collection were overseen by trained nurses who were briefed on standardized procedures. All preanalytical, analytical, and postanalytical processes adhered to standard operating procedures. The laboratory followed quality management practices aligned with accreditation requirements. Internal quality control was maintained using both normal and pathological quality control materials.

Data Analysis

Data were entered and analyzed using SPSS version 25. Descriptive statistics were used to summarize sociodemographic characteristics and laboratory values. To assess differences between admission and discharge values, the Wilcoxon signed-rank test was applied for nonparametric data. The Bland–Altman method was used to evaluate the agreement between admission and discharge measurements for parameters without statistically significant differences. Repeated measures analysis of variance (ANOVA) was employed to assess mean differences across multiple time points or conditions. Post hoc analyses were conducted to identify specific categories with significant differences. A p-value of <0.05 was considered statistically significant.

Ethical Approval and Informed Consent

Ethical clearance was obtained from the Research and Ethics Review Committee of the Department of Medical Laboratory Sciences, Addis Ababa University (Protocol No. DRERC/583/21/MLS). Prior to data collection, permission was granted by the Ministry of Health and St. Peter Specialized Hospital. Written informed consent was obtained from each participant after explaining the study's purpose and procedures. Participant confidentiality was strictly maintained throughout the study.

Results

Sociodemographic Characteristics of the Study

Participants: A total of 162 patients were initially enrolled in this study. However, data from six individuals were excluded due to death ($n = 4$) or referral to other healthcare facilities for further management ($n = 2$). Consequently, 156 participants were included in the final analysis. Of these, 96 (61.5%) were male. The mean age of participants was 55.46 ± 14.32 years. The majority (80.4%) were over 41 years of age. Most participants were married (70.5%, $n = 110$), and a smaller proportion (18.6%, $n = 29$) were from rural areas (Supplementary Table 1).

Changes in Lipid, Liver, and Renal Function Tests at Admission and Discharge:

Significant differences were observed in several biochemical parameters between admission and discharge. Using the nonparametric Wilcoxon signed-rank test, statistically significant increases were noted in total cholesterol, high-density lipoprotein (HDL), low-density lipoprotein (LDL), and albumin levels at discharge compared to admission ($p < 0.05$). Conversely, alanine aminotransferase (ALT) levels were significantly higher at admission ($p = 0.001$). No significant differences were found in creatinine or urea levels (Table 1).

Table 1: Paired analysis of lipid profiles, liver enzymes, and renal function tests at admission and discharge ($n = 156$)

Parameter	Admission (Median $\pm$ SD)	Discharge (Median $\pm$ SD)	Wilcoxon p-value
Total cholesterol (mg/dL)	125.05 $\pm$ 43.86	150.60 $\pm$ 48.91	<0.001*
HDL (mg/dL)	29.55 $\pm$ 12.90	34.10 $\pm$ 15.11	<0.001*
LDL (mg/dL)	61.70 $\pm$ 32.18	80.85 $\pm$ 35.95	<0.001*
Triglycerides (mg/dL)	137.05 $\pm$ 79.17	151.05 $\pm$ 80.18	0.742
Albumin (g/L)	26.30 $\pm$ 6.29	27.85 $\pm$ 6.45	0.003*
AST (U/L)	31.00 $\pm$ 34.51	30.40 $\pm$ 28.07	0.339
ALT (U/L)	26.80 $\pm$ 34.94	18.40 $\pm$ 39.17	0.001*
Direct Bilirubin (mg/dL)	0.15 $\pm$ 0.21	0.15 $\pm$ 0.20	0.465
Total Bilirubin (mg/dL)	0.27 $\pm$ 0.28	0.28 $\pm$ 2.16	0.456
Creatinine (mg/dL)	0.72 $\pm$ 0.63	0.74 $\pm$ 0.67	0.864
Urea (mg/dL)	32.90 $\pm$ 32.58	32.35 $\pm$ 24.94	0.999

*Significant at $p < 0.05$

Abbreviations: HDL- high-density lipoprotein, LDL; low-density lipoprotein, AST; aspartate aminotransferase, ALT; alanine aminotransferase TG; triglyceride, g/dl; gram per decilitre, mg/dl; milligram per decilitre, U/L; international unit per Liter

*P values indicate the differences between admission and discharge values (median discharge-median admission). $P < 0.05$ was considered to indicate statistical significance.

Bland–Altman plots further confirmed the agreement of most parameters between admission and discharge. Only triglycerides and AST showed notable variation, with 5.8% and 5.1% of values, respectively, falling outside the

95% limits of agreement. Most values for creatinine, urea, and total bilirubin remained within the limits (Figures 1A–1E).

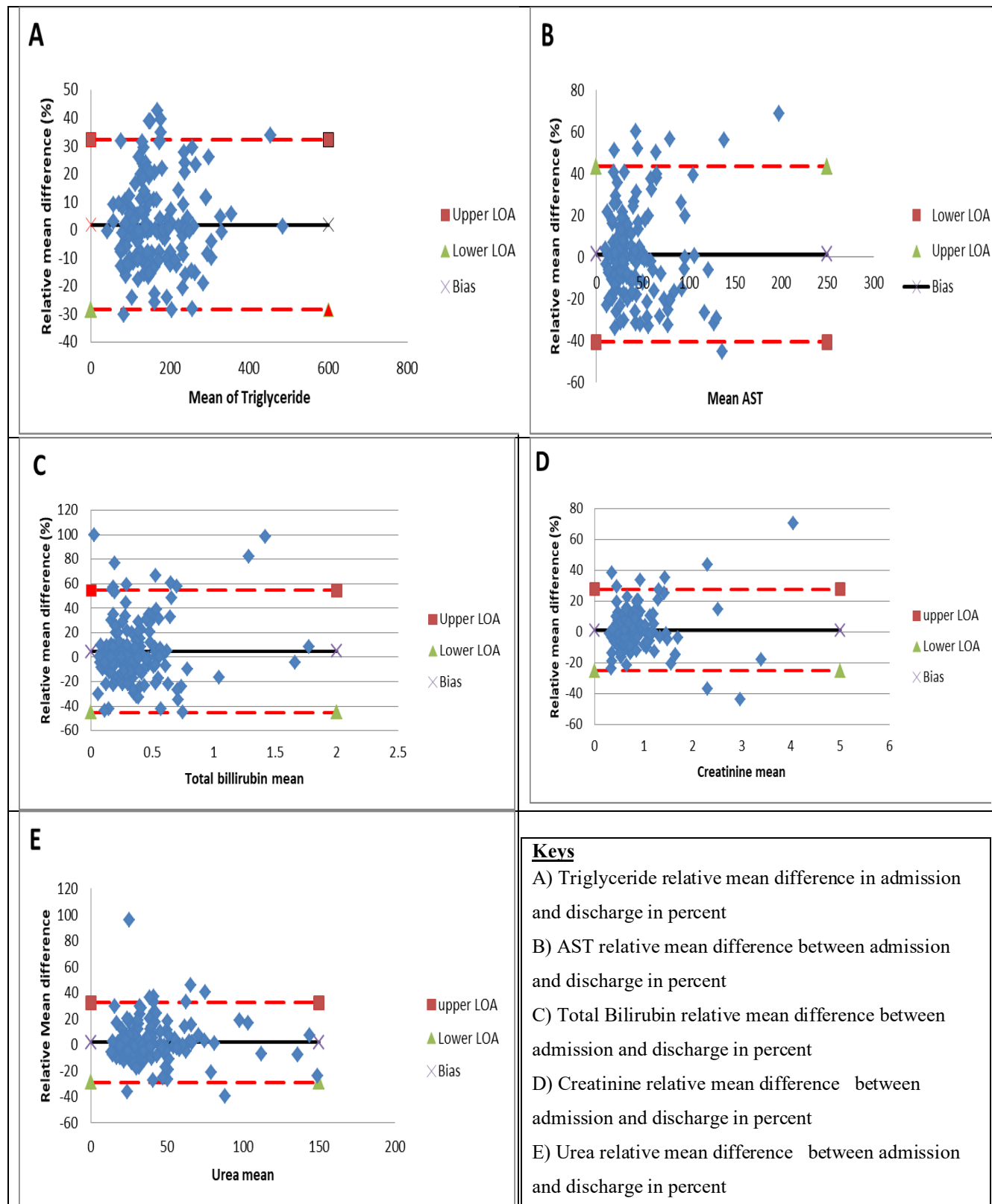


Figure 1: Bland Altman plot of relative mean differences between admission and discharge in some laboratory profile values

*Lipid, liver, and renal changes in severe COVID-19 patients***Factors Influencing Admission Laboratory Values**

Analysis of variance (ANOVA) was performed to assess the influence of age and sex on admission laboratory values.

- **Gender:** Among all measured parameters, only ALT levels differed significantly between males

and females, with higher levels observed in males ($F = 7.920$, $p = 0.006$).

- **Age Groups:** One-way ANOVA revealed significant differences in ALT, albumin, direct bilirubin, total bilirubin, and urea across age categories ($p < 0.05$) (Table 2).

Table 2: One-way ANOVA of laboratory test results at admission by age group and gender

Dependent variable	Age groups and Gender category	Mean	95% CI for Mean		F test	p value
			lower	upper		
Total cholesterol	< 30 years (n=8)	155.0625	115.9204	194.2046	2.146	0.097
	30-40 years (n=22)	144.1318	127.3701	160.8936		
	41-60 years (n=63)	137.5873	126.3422	148.8324		
	>60years (n=63)	124.6571	113.7170	135.5973		
HDL	< 30 years(n=8)	28.8250	14.9295	42.7205	0.109	0.954
	30-40 years (n=22)	31.5045	26.6542	36.3549		
	41-60 years (n=63)	30.9810	27.3769	34.5850		
	>60years (n=63)	30.3365	27.3704	33.3026		
LDL	< 30 years (n=8)	72.7500	43.3835	102.1165	1.049	0.373
	30-40 years (n=22)	73.0864	58.8860	87.2867		
	41-60 years (n=63)	66.8857	58.4526	75.3188		
	>60years (n=63)	60.7048	53.0290	68.3805		
Triglyceride	< 30 years (n=8)	163.5500	97.0764	230.0236	1.499	0.217
	30-40 years (n=22)	148.8818	117.3398	180.4239		
	41-60 years (n=63)	175.8857	154.6410	197.1304		
	>60years (n=63)	147.9127	128.9418	166.8836		
AST	< 30 years(n=8)	70.8375	18.3459	123.3291	1.714	0.166
	30-40 years (n=22)	36.4455	24.7953	48.0956		
	41-60 years (n=63)	42.0727	31.8911	52.2543		
	>60years (n=63)	43.7016	35.2720	52.1312		
ALT	< 30 years(n=8)	71.2375	2.1264	140.3486	3.512	0.017*
	30-40 years (n=22)	33.7818	14.4497	53.1140		
	41-60 years (n=63)	32.6632	22.6628	42.6636		
	>60years (n=63)	25.0854	18.9448	31.2260		
Albumin	< 30 years(n=8)	27.5875	20.8972	34.2778	3.088	0.029*
	30-40 years (n=22)	29.7818	27.3414	32.2222		
	41-60 years (n=63)	25.9048	24.2444	27.5651		
	>60years (n=63)	25.3000	23.8661	26.7339		
Direct bilirubin	< 30 years(n=8)	0.6100	-0.1630	1.3830	5.704	0.001*
	30-40 years (n=22)	0.1619	0.1095	0.2142		
	41-60 years (n=63)	0.2279	0.1589	0.2969		
	>60years (n=63)	0.1918	0.1599	0.2237		
Bilirubin total	< 30 years (n=8)	0.6524	0.1098	1.1950	3.800	0.012*
	30-40 years (n=22)	0.3172	0.2132	0.4213		
	41-60 years (n=63)	0.3067	0.2381	0.3754		
	>60years (n=63)	0.3441	0.2906	0.3975		
Creatinine	< 30 years (n=8)	0.7925	0.3684	1.2166	0.328	0.805
	30-40 years (n=22)	0.7382	0.6055	0.8709		
	41-60 years (n=63)	0.8608	0.6703	1.0513		
	>60years (n=63)	0.8859	0.7387	1.0330		
Urea	< 30 years (n=8)	26.3288	11.7580	40.8995	2.364	0.073
	30-40 years (n=22)	31.3727	24.1018	38.6437		
	41-60 years (n=63)	40.9270	32.7810	49.0730		
	>60years (n=63)	48.6810	39.3816	57.9803		
Total cholesterol	Male (n=96)	133.7708	124.8125	142.7292	0.022	0.882
	Female (n=60)	134.8467	123.5752	146.1182		
HDL	Male (n=96)	29.8448	27.4629	32.2267	1.056	0.306
	Female (n=60)	32.0267	28.2673	35.7861		

LDL	Male (n=96)	65.7708	59.1767	72.3650	0.010	0.920
	Female (n=60)	65.2350	57.0030	73.4670		
Triglyceride	Male (n=96)	167.4531	150.6982	184.2081	2.141	0.145
	Female (n=60)	148.4600	129.7798	167.1402		
AST	Male (n=96)	45.6029	37.5123	53.6936	0.843	0.360
	Female (n=60)	39.9067	31.1741	48.6392		
ALT	Male (n=96)	38.5654	29.2725	47.8583	7.920	0.006*
	Female (n=60)	20.8163	15.3591	26.2735		
Albumin	Male (n=96)	25.7646	24.5619	26.9673	1.772	0.185
	Female (n=60)	27.1400	25.3844	28.8956		
Direct bilirubin	Male (n=96)	0.2323	0.1665	0.2981	0.218	0.641
	Female (n=60)	0.2097	0.1493	0.2701		
Total bilirubin	Male (n=96)	0.3464	0.2905	0.4024	0.092	0.762
	Female (n=60)	0.3323	0.2562	0.4084		
Creatinine	Male (n=96)	0.9171	0.7692	1.0650	2.891	0.091
	Female (n=60)	0.7430	0.6421	0.8439		
Urea	Male (n=96)	45.0430	38.0941	51.9919	2.250	0.136
	Female (n=60)	37.0333	29.4831	44.5836		

* Indicates that the significance level was less than 0.05.

Post hoc analysis identified that:

- **ALT and AST:** Participants aged <30 years had significantly higher ALT and AST levels than those aged 30–60 and >60 years ($p < 0.05$).
- **Albumin:** Albumin levels were significantly higher in the 30–40 age group than in those aged 41–60 and >60 ($p < 0.05$).
- **Direct and Total Bilirubin:** Significantly higher levels were observed in the <30 age group and among those aged 30–40 compared to older participants.
- **Urea:** The <30 age group had significantly lower urea levels than those aged >60 years (mean difference = -17.31; 95% CI: -33.04 to -1.58; $p = 0.031$) (Table 3).

Table 3: Post hoc comparisons of selected lab parameters across age groups (only significant comparisons shown)

Parameter	Age Groups Compared	Mean Difference	95% CI	p-value
AST	<30 vs 30–40	34.39	3.87 to 64.92	0.027
	<30 vs 41–60	28.76	1.01 to 56.52	0.042
ALT	<30 vs 30–40	37.46	6.26 to 68.66	0.019
	<30 vs 41–60	38.57	10.21 to 66.94	0.008
	<30 vs >60	46.15	17.79 to 74.52	0.002
Albumin	30–40 vs 41–60	3.88	0.86 to 6.90	0.012
	30–40 vs >60	4.48	1.46 to 7.50	0.004
Direct Bilirubin	<30 vs 30–40	0.45	0.22 to 0.68	<0.001
Total Bilirubin	<30 vs 30–40	0.34	0.11 to 0.56	0.004
Urea	30–40 vs >60	-17.31	-33.04 to -1.58	0.031

Discussion

This follow-up study investigated the impact of severe COVID-19 infection on common laboratory parameters, specifically lipid profiles, liver function tests, and renal function markers, among hospitalized patients. A key finding was that total cholesterol, high-density lipoprotein cholesterol (HDL-C), and low-density lipoprotein cholesterol (LDL-C) levels were significantly lower at admission compared to discharge. These observations align with previous reports from France and China, which have documented hypolipidemia in patients with severe or critical COVID-19 [Wei et al., 2020; Fan J, 2020]. Several studies globally have also shown that reduced lipid levels, particularly HDL-C and LDL-C, are

associated with the severity of COVID-19 illness (Malik et al. 2021; Hu et al. 2020; Qin et al. 2020; Zhu et al. 2020; Liu W et al. 2020).

The development of hypolipidemia in COVID-19 patients may be linked to systemic inflammation and liver dysfunction. Proinflammatory cytokines such as TNF- α and IL-6, which are elevated during cytokine storms, have been implicated in disrupting lipid metabolism by inhibiting hepatic lipid synthesis, increasing lipid consumption, and impairing cholesterol transport (Liu et al. 2020; Malik et al. 2021; Lopes et al. 2018).

Unlike cholesterol levels, triglyceride (TG) levels did not show a statistically significant change between admission

and discharge in our cohort, consistent with the findings of Hu et al. [2020]. However, this contrasts with reports indicating that elevated TG levels may be a marker of disease severity or poor prognosis in COVID-19 patients (Changaripour et al. 2021; Wei et al. 2020). The discrepancy may be attributed to differences in study design, sample characteristics, or disease severity. It has been hypothesized that increased inflammatory cytokines in severe COVID-19 cases can lead to excess free fatty acid production and elevated TG synthesis (Sun et al. 2020; Mohammedsaeed et al. 2020). These observations highlight the need for further investigation in diverse patient populations.

This study also assessed the effects of COVID-19 on liver function parameters. While both ALT and AST levels were elevated at admission compared to discharge, only ALT showed a statistically significant reduction over time. Elevated liver enzymes during COVID-19 infection have been previously linked to direct viral hepatotoxicity, systemic inflammation, and hypoxic injury (Parohan et al. 2020; Bloom et al. 2021; De la Rica et al. 2020; Aziz et al. 2020). AST, however, is less liver-specific and may reflect systemic tissue injury rather than isolated liver dysfunction (Bertolini et al. 2020).

Serum albumin levels were significantly lower at admission, a finding commonly associated with systemic inflammation, malnutrition, and severe illness. Hypoalbuminemia has also been identified as a predictor of poor outcomes in severe COVID-19 cases (Aziz et al. 2020; Vancsa et al. 2020).

It is important to note that abnormal liver function test results in COVID-19 may also be influenced by pre-existing liver disease or other comorbidities (Zhang et al. 2020). In our study, younger patients (<30 years) had significantly higher ALT levels at admission compared to older age groups. This finding contrasts with previous reports that have associated elevated ALT levels with older age and more severe disease (Xu et al. 2020). The differences may be due to sample size variations, study settings, or other confounding factors, warranting further exploration.

Study Limitations

This study has several limitations. First, we were unable to include data from deceased patients due to small sample size and missing follow-up data. Second, the laboratory investigations were limited in scope and did not include more advanced biomarkers of liver or renal function. Additionally, potential confounding factors such as medication use (including traditional remedies) or pre-existing comorbidities were not comprehensively assessed. Future studies with larger cohorts and broader diagnostic panels are recommended to validate and expand upon these findings.

Conclusion

This study demonstrated that in patients with severe COVID-19, levels of total cholesterol, HDL-C, LDL-C, and albumin were significantly lower at hospital admission compared to discharge, while ALT levels were significantly higher at admission. These changes suggest that SARS-CoV-2 infection leads to notable metabolic and hepatic alterations, particularly during the acute phase of illness. Furthermore, younger age and male sex were associated with elevated ALT levels, and renal markers such as urea were significantly influenced by age and gender.

These findings underscore the importance of monitoring lipid and liver function parameters during the clinical management of severe COVID-19. Regular assessment of these biomarkers may aid in evaluating disease progression, guiding treatment decisions, and improving patient outcomes. Integrating lipid and hepatic function monitoring into COVID-19 care protocols could enhance the quality and effectiveness of clinical management, particularly in resource-limited settings.

Conflict of Interest

The authors declare that they have no conflicts of interest related to this study.

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