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Impact of COVID-19 on Organ Function in Individuals with Type 2 Diabetes Mellitus: A Biochemical Analysis of Kidney, Liver, and Lipid Profiles

Olukemi Daodu [1]; Abolape Iyanda [2] *
1.Department of Chemical Pathology, University College Hospital, Ibadan
2. Department of Chemical Pathology Ladoke Akintola University of Technology Ogbomosho

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ABSTRACT: *This study investigates the impact of COVID-19 and Type 2 Diabetes Mellitus (T2DM) on organ function, focusing on kidney and liver function and lipid metabolism. Chronic hyperglycemia in diabetes predisposes patients to a pro-inflammatory and pro-thrombotic state, exacerbating COVID-19-related complications. This cross-sectional study involved 400 participants divided into five groups: T2DM without COVID-19, T2DM patients recently recovered from COVID-19, T2DM patients three months post-COVID-19 recovery, COVID-19-recovered individuals without T2DM, and apparently healthy controls. Biochemical markers for kidney function (urea, creatinine, uric acid), liver function (AST, ALT, albumin), and lipid profile (total cholesterol, triglycerides, HDL, LDL) were analyzed using enzymatic, colorimetric, and spectrophotometric methods. Fasting plasma glucose (FPG) and hemoglobin A1c (HbA1c) were also measured to assess glycemic control.The results revealed significant alterations in kidney function markers, particularly elevated urea ($F = 49.4$, $p < 0.001$) and creatinine ($F = 89.1$, $p < 0.001$) levels in T2DM patients recovering from COVID-19. Liver function markers showed increased AST ($F = 142.2$, $p < 0.001$) and ALT ($F = 200.6$, $p < 0.001$) levels with reduced albumin ($F = 264.5$, $p < 0.001$), indicating hepatic stress in this group. Dyslipidemia, characterized by elevated total cholesterol ($F = 103.3$, $p < 0.001$) and triglycerides ($F = 69.5$, $p < 0.001$) and reduced HDL ($F = 47.8$, $p < 0.001$), was prominent among T2DM participants recovering from COVID-19. Pearson's correlation analysis demonstrated strong positive associations between fasting glucose levels and markers of kidney (urea: $r = 0.352$, $p < 0.001$; creatinine: $r = 0.208$, $p < 0.001$) and liver dysfunction (AST: $r = 0.439$, $p < 0.001$; ALT: $r = 0.496$, $p < 0.001$), as well as lipid abnormalities (total cholesterol: $r = 0.172$, $p = 0.001$; triglycerides: $r = 0.321$, $p < 0.001$).*
These findings highlight the compounded impact of COVID-19 and T2DM on organ function, emphasizing the need for vigilant monitoring and tailored management strategies for affected individuals. Understanding the biochemical interplay between these conditions is critical for mitigating long-term complications and improving outcomes in high-risk populations. This study underscores the importance of early detection and intervention to address the multifaceted effects of COVID-19 on individuals with T2DM.

Keywords: Type 2 Diabetes Mellitus, COVID-19, Inflammatory response

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INTRODUCTION

The COVID-19 pandemic has brought significant global health challenges, especially for individuals with comorbidities such as diabetes mellitus (DM). Diabetes mellitus, particularly Type 2 Diabetes Mellitus (T2DM), is associated with a higher risk of severe outcomes in COVID-19, including increased mortality and complications affecting organ function (Bornstein et al., 2020). The intersection of these two conditions creates a complex interplay of pathophysiological mechanisms that exacerbate pre-existing metabolic and inflammatory states, leading to heightened vulnerability in affected individuals (Apicella et al., 2020).

COVID-19 primarily targets the respiratory system, often leading to pneumonia and acute respiratory distress syndrome (ARDS). However, emerging evidence underscores its ability to affect multiple organ systems, a phenomenon linked to both direct viral invasion and systemic inflammation. Notably, the kidneys, liver, cardiovascular system, and endocrine pathways are frequently implicated, suggesting that SARS-CoV-2 has a broader tropism than initially understood (Gupta et al., 2020). This systemic impact is particularly concerning for diabetic patients, who are already predisposed to chronic organ damage. Chronic hyperglycemia, a hallmark of diabetes mellitus, fosters a pro-inflammatory state by promoting the activation of nuclear factor kappa B (NF-κB) pathways and increasing oxidative stress. These changes not only heighten the baseline inflammation but also amplify the cytokine storm commonly observed in severe COVID-19 cases, potentially exacerbating systemic and organ-specific complications. Moreover, a pro-thrombotic state induced by hyperglycemia contributes to microvascular and macrovascular complications, which can worsen the impact of SARS-CoV-2 on organs such as the kidneys and heart. This dual vulnerability raises the risks of acute kidney injury (AKI), liver dysfunction, and cardiovascular complications in diabetic patients infected with COVID-19 (Huang et al., 2020; Zhou et al., 2020).

The interaction between diabetes and COVID-19 involves complex mechanisms, one of which is the angiotensin-converting enzyme 2 (ACE2) receptor. ACE2 serves as a critical viral entry point for SARS-CoV-2, facilitating its attachment and entry into host cells. In diabetic individuals, ACE2 expression is dysregulated, with alterations influenced by the use of medications such as ACE inhibitors and angiotensin receptor blockers (ARBs) (Yang et al., 2020). While these medications may offer protective cardiovascular effects, they could inadvertently increase the susceptibility of certain cells, including those in the lungs, kidneys, and heart, to SARS-CoV-2 infection. Beyond its role in viral entry, ACE2 is integral to the renin-angiotensin-aldosterone system (RAAS), which regulates blood pressure, fluid balance, and inflammatory responses. Dysregulation of this pathway in diabetic patients could exacerbate COVID-19-induced tissue damage. Furthermore, the systemic inflammation and hypercoagulability associated with both conditions create a feedback loop that worsens organ dysfunction. This interaction underscores the importance of personalized therapeutic approaches, including the careful management of glycemic

levels and the judicious use of RAAS-modulating drugs, to mitigate the compounded effects of diabetes and COVID-19 on organ health.

Understanding the combined effects of COVID-19 and diabetes on organ function is crucial for tailoring interventions and improving patient outcomes. This study aims to evaluate the influence of COVID-19 on organ function in individuals with T2DM, considering the interplay of biochemical markers and inflammatory responses during recovery. Such insights will contribute to the growing body of evidence required for managing these interconnected health challenges effectively.

MATERIALS AND METHODS

Study Design
This was a multicentre cross-sectional study, female and male subjects aged between 30 and 60 years, who met the inclusion criteria were recruited for the study.

Study Location
For this study, 3 centers were used, Covid-19 Centre in University College Hospital Ibadan, Virology Department College of Medicine University of Ibadan, and Infectious Disease Centre Olodo Ibadan, all in Nigeria.

Study Population
The study population consists of 400 participants, divided into five distinct groups. These groups include 80 T2DM subjects without a history of COVID-19 (positive controls), 80 COVID-19 recovered T2DM subjects at the onset of recovery (0 Month), 80 COVID-19 recovered T2DM subjects three months post-recovery (3 Month), 80 COVID-19 recovered subjects without T2DM, and 80 apparently healthy individuals (negative controls). The participants, aged between 30 and 60 years, include both male and female subjects in equal proportion across all groups. This demographic diversity ensures a robust comparison of biochemical markers, accounting for the influence of both gender and the presence of T2DM in the context of COVID-19 recovery.

Ethical Considerations
Ethical approval was obtained from Ministry of Health, Oyo State Ethical Review Committee Secretariat Ibadan. Informed consent forms were filled by the participants. All information obtained from the participants were kept in strict confidentiality.

Biochemical Analysis
Biochemical analysis in the study was performed using standard laboratory techniques to evaluate kidney function, liver function, lipid profiles, and glycemic control. Kidney function markers, including urea, creatinine, and uric acid, were measured using enzymatic colorimetric methods. These assays rely on specific enzymatic reactions to quantify metabolite concentrations accurately.

For liver function assessment, aspartate transaminase (AST) and alanine transaminase (ALT) levels were determined through spectrophotometric enzyme activity assays, which measure the conversion of specific substrates by these enzymes. Albumin was analyzed using the bromocresol green dye-binding method, a widely accepted technique for protein quantification. The lipid profile, including total cholesterol, triglycerides, high-density lipoprotein (HDL), and low-density lipoprotein (LDL), was assessed using enzymatic methods. For glycemic control, fasting plasma glucose (FPG) was analyzed using the glucose oxidase-peroxidase method while Hemoglobin A1c (HbA1c) was quantified using high-performance liquid chromatography (HPLC).

Statistical Analysis

Statistical analysis was performed using the Statistical Package for Social Sciences (SPSS) software version 28.0. One-way ANOVA with post-hoc Tukey's tests was used to compare the various groups in the study. Pearson's correlation test was used to analyze for relationships between variables. Level of statistical significance was set at $p < 0.05$.

RESULT

Table 1 presents a comparison of kidney function markers among different participant groups. For Urea levels, Group 5 had the lowest mean, while Group 4 showed a lower mean (33.4±7.6 mg/dl) compared to Groups 1 (39.7±3.3 mg/dl) and 2 (41.3±8.5 mg/dl). Creatinine levels varied significantly, with Group 5 having the lowest mean at 0.9±0.3 mg/dl. Group 4 exhibited a lower Creatinine mean (0.9±0.2 mg/dl) compared to Groups 1 (1.2±0.3 mg/dl) and 2 (1.5±0.3 mg/dl). Uric Acid levels also differed significantly among the groups. Group 5 displayed the lowest mean at 5.3±1.1 mg/dl, and Group 2 showed a lower mean (6.4±1.3 mg/dl) compared to Groups 1 (8.9±0.8 mg/dl) and 4 (8.4±1.4 mg/dl).

Table 2 outlines a comparison of liver function markers among different participant groups, focusing on the impact of Type 2 Diabetes Mellitus (DM) and COVID-19 infection. AST levels varied significantly across the groups, with Group 5 exhibiting the lowest mean at 28.1±12.1 IU/l. Both Group 2 (48.6±6.2 IU/l) and Group 4 (49.1±6.1 IU/l) showed lower AST means compared to Group 1 (50.9±6.1 IU/l). ALT levels also demonstrated significant differences across the 4 groups, with Group 5 displaying the lowest mean at 25.9±12.2 IU/l. Both Group 2 (47.6±6.3 IU/l) and Group 4 (49.7±4.4 IU/l) showed lower ALT means compared to Group 1 (49.8±2.9 IU/l). Albumin levels varied significantly among the groups, with Group 5 displaying the highest mean at 4.4±0.7 g/dl. Both Group 2 (3.2±0.4 g/dl) and Group 1 (2.8±0.5 g/dl) showed lower Albumin means compared to Group 4 (2.6±0.2 g/dl).

Table 3 illustrates a comparison of lipid profile levels among different participant groups, focusing on the influence of Type 2 Diabetes Mellitus (DM) and COVID-19 infection. Total Cholesterol levels varied significantly across groups, with Group 2 exhibiting the highest mean at 210.6±14.6 mg/dl. Both Group 4 (142.9±16.9 mg/dl) and Group 5 (161.2±31.5 mg/dl) showed lower Total

Cholesterol means compared to Group 1 (185.0±34.5 mg/dl). Triglyceride levels also demonstrated significant differences, with Group 2 displaying the highest mean at 156.5±23.2 mg/dl. Both Group 4 (118.2±18.5 mg/dl) and Group 5 (89.3±15.6 mg/dl) showed lower Triglyceride means compared to Group 1 (120.3±48.5 mg/dl). HDL levels varied significantly among the groups, with Group 4 displaying the lowest mean at 32.1±6.3 mg/dl. Group 1 (41.4±5.8 mg/dl) and Group 5 (41.4±7.3 mg/dl) showed higher HDL means compared to Group 2 (40.7±3.4 mg/dl). LDL levels also demonstrated significant differences among groups, with Group 2 displaying the highest mean at 148.2±25.7 mg/dl. Both Group 4 (92.2±15.9 mg/dl) and Group 5 (103.9±24.9 mg/dl) showed lower LDL means compared to Group 1 (119.9±28.8 mg/dl).

Table 4 presents the Pearson's correlation coefficients between Fasting Plasma Glucose (FPG) and various biochemical markers in the study population. FPG showed positive correlations with Total Cholesterol ($r = 0.172$, $p = 0.001$), Triglyceride ($r = 0.321$, $p < 0.001$), Low-Density Lipoprotein (LDL) ($r = 0.181$, $p < 0.001$), Urea ($r = 0.352$, $p < 0.001$), Creatinine ($r = 0.208$, $p < 0.001$), Uric Acid ($r = 0.357$, $p < 0.001$), Aspartate Aminotransferase (AST) ($r = 0.439$, $p < 0.001$), and Alanine Aminotransferase (ALT) ($r = 0.496$, $p < 0.001$). Conversely, a negative correlation was observed between FPG and High-Density Lipoprotein (HDL) ($r = -0.050$, $p = 0.317$) as well as Albumin ($r = -0.518$, $p < 0.001$).

Table 5 illustrates the Pearson's correlation between the marker of glycemic control, Hemoglobin A1c (HbA1c), the various biochemical markers in the study population. HbA1c showed positive correlations with Total Cholesterol ($r = 0.442$, $p < 0.001$), Triglyceride ($r = 0.546$, $p < 0.001$), Low-Density Lipoprotein (LDL) ($r = 0.426$, $p < 0.001$), Urea ($r = 0.257$, $p < 0.001$), Creatinine ($r = 0.335$, $p < 0.001$), Aspartate Aminotransferase (AST) ($r = 0.373$, $p < 0.001$), and Alanine Aminotransferase (ALT) ($r = 0.375$, $p < 0.001$). Conversely, a negative correlation was observed between HbA1c and High-Density Lipoprotein, though not significant ($r = 0.084$, $p = 0.092$) but significant for Albumin ($r = -0.449$, $p < 0.001$).

TABLE 1: COMPARISON OF KIDNEY FUNCTION MARKERS AMONG PARTICIPANTS WITH AND WITHOUT TYPE 2 DM INFECTED WITH COVID-19 AND CONTROLS

MARKERS	GROUP 1 Mean±SD	GROUP 2 Mean±SD	GROUP 4 Mean±SD	GROUP 5 Mean±SD	F-value	p-value
Urea (mg/dl)	39.7±3.3	41.3±8.5$^{\$}$	33.4±7.6$^{\#\$\wedge}$	28.2±9.6$^{\#}$	49.4	<0.001*
Creatinine (mg/dl)	1.2±0.3	1.5±0.3$^{\#\$}$	0.9±0.2$^{\#\wedge}$	0.9±0.3$^{\#}$	89.1	<0.001*
Uric acid (mg/dl)	8.9±0.8	6.4±1.3$^{\#\$}$	8.4±1.4$^{\#\$\wedge}$	5.3±1.1$^{\#}$	156.9	<0.001*

KEYS
Group 1 – Type 2 DM patients without history of Covid-19 (Positive controls)
Group 2 - Type 2 DM patients that recently recovered from Covid-19

Group 4 – Covid-19 patients without history of Type 2 DM
Group 5 - Apparently healthy participants (Negative controls)
*Significant at $p < 0.05$
[#]Significantly different from group 1
[$]Significantly different from group 5
^Significantly different from group 2
Abbreviations: SD – standard deviation

TABLE 2: COMPARISON OF LIVER FUNCTION MARKERS AMONG PARTICIPANTS WITH AND WITHOUT TYPE 2 DM INFECTED WITH COVID-19 AND CONTROLS

MARKERS	GROUP 1 Mean±SD	GROUP 2 Mean±SD	GROUP 4 Mean±SD	GROUP 5 Mean±SD	F-value	p-value
AST (IU/l)	50.9±6.1	48.6±6.2[$]	49.1±6.1[$]	28.1±12.1[#]	142.2	<0.001*
ALT (IU/l)	49.8±2.9	47.6±6.3[#$]	49.7±4.4[$]	25.9±12.2[#]	200.6	<0.001*
Albumin (g/dl)	2.8±0.5	3.2±0.4[#$]	2.6±0.2[#$^]	4.4±0.7[#]	264.5	<0.001*

KEYS
Group 1 – Type 2 DM patients without history of Covid-19 (Positive controls)
Group 2 - Type 2 DM patients that recently recovered from Covid-19
Group 4 – Covid-19 patients without history of Type 2 DM
Group 5 - Apparently healthy participants (Negative controls)
*Significant at $p < 0.05$
[#]Significantly different from group 1
[$]Significantly different from group 5
^Significantly different from group 2
Abbreviations: SD – standard deviation; AST – Aspartate transaminase; ALT – Alanine transaminase

TABLE 3: COMPARISON OF LIPID PROFILE LEVELS AMONG PARTICIPANTS WITH AND WITHOUT TYPE 2 DM INFECTED WITH COVID-19 AND CONTROLS

MARKERS	GROUP 1 Mean±SD	GROUP 2 Mean±SD	GROUP 4 Mean±SD	GROUP 5 Mean±SD	F-value	p-value
Total cholesterol (mg/dl)	185.0±34.5	210.6±14.6[#$]	142.9±16.9[#$^]	161.2±31.5[#]	103.3	<0.001*
Triglyceride (mg/dl)	120.3±48.5	156.5±23.2[#$]	118.2±18.5[$^]	89.3±15.6[#]	69.5	<0.001*
HDL (mg/dl)	41.4±5.8	40.7±3.4	32.1±6.3[#$^]	41.4±7.3	47.8	<0.001*
LDL (mg/dl)	119.9±28.8	148.2±25.7[#$]	92.2±15.9[#$^]	103.9±24.9[#]	79.6	<0.001*

KEYS

KEYS
Group 1 – Type 2 DM patients without history of Covid-19 (Positive controls)
Group 2 - Type 2 DM patients that recently recovered from Covid-19
Group 4 – Covid-19 patients without history of Type 2 DM
Group 5 - Apparently healthy participants (Negative controls)
*Significant at $p < 0.05$
#Significantly different from group 1
$Significantly different from group 5
^Significantly different from group 2
Abbreviations: SD – standard deviation; HDL – High density lipoproteins; LDL – Low density lipoproteins

TABLE 4: RELATIONSHIP BETWEEN FASTING PLASMA GLUGOSE AND INFLAMMATORY AND OTHER BIOCHEMICAL MARKERS IN THE STUDY POPULATION (PEARSONS' CORRELATION)

MARKERS	r-value	p-value
Total Chol. (mg/dl)	0.172	0.001*
Triglyceride (mg/L)	0.321	<0.001*
HDL (mg/dl)	-0.050	0.317
LDL (mg/dl)	0.181	<0.001*
Urea (mg/dl)	0.352	<0.001*
Creatinine (mg/dl)	0.208	<0.001*
Uric acid (mg/dl)	0.357	<0.001*
AST (IU/L)	0.439	<0.001*
ALT (IU/L)	0.496	<0.001*
Albumin (g/dl)	-0.518	<0.001*

*Significant at $p < 0.05$

TABLE 5: RELATIONSHIP BETWEEN MARKER OF GLYCEMIC CONTROL (HbA1c) AND INFLAMMATORY AND OTHER BIOCHEMICAL MARKERS IN THE STUDY POPULATION (PEARSONS' CORRELATION)

MARKERS	r-value	p-value
Total Chol. (mg/dl)	0.442	<0.001*
Triglyceride (mg/L)	0.546	<0.001*
HDL (mg/dl)	-0.084	0.092
LDL (mg/dl)	0.426	<0.001*
Urea (mg/dl)	0.257	<0.001*
Creatinine (mg/dl)	0.335	<0.001*
Uric acid (mg/dl)	0.030	0.555
AST (IU/L)	0.373	<0.001*
ALT (IU/L)	0.375	<0.001*
Albumin (g/dl)	-0.449	<0.001*

*Significant at $p < 0.05$

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DISCUSSION

The observed alterations in kidney function markers, particularly the increased levels of urea and creatinine, among individuals with Type 2 Diabetes Mellitus (T2DM) recovering from COVID-19 underscore the complex interplay between the viral infection and renal health. These findings align with emerging evidence suggesting that COVID-19 is associated with renal complications, adding an additional layer of concern for individuals with pre-existing metabolic conditions.

Several studies have reported a high incidence of acute kidney injury (AKI) in COVID-19 patients, with increased risks of severe outcomes and mortality (Cheng et al., 2020). The elevation in urea levels may indicate impaired renal filtration and excretion, suggesting potential stress on the kidneys. Similarly, the rise in creatinine levels is indicative of compromised glomerular filtration rate, emphasizing the need for vigilant monitoring of renal function in individuals with T2DM during the recovery phase from COVID-19.

The impact on kidney function may be attributed to several mechanisms. First, the direct cytopathic effect of the virus on renal cells has been proposed. Studies have detected the presence of severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) in renal tissues, suggesting viral tropism to the kidneys (Cheng et al., 2020). This viral invasion may induce inflammation and damage to renal cells, contributing to the observed alterations in kidney function markers.

Second, the systemic inflammatory response induced by COVID-19 is known to play a pivotal role in renal dysfunction. Elevated levels of proinflammatory cytokines, such as interleukin-6 (IL-6), have been reported in COVID-19 patients (Chen et al., 2020). This inflammatory milieu can lead to endothelial dysfunction, microvascular thrombosis, and oxidative stress in the kidneys, exacerbating the risk of AKI in susceptible individuals.

The observed kidney function alterations in individuals recovering from COVID-19 with T2DM highlight the intricate relationship between the viral infection and metabolic comorbidities. Moreover, these findings emphasize the need for tailored management strategies that address both the viral infection and the potential exacerbation of pre-existing conditions. Regular monitoring of kidney function, especially in high-risk populations, is imperative for early detection and intervention to mitigate long-term renal complications.

In the same vein, the impact of COVID-19 on liver function, as evidenced by the results of liver function markers in individuals with Type 2 Diabetes Mellitus (T2DM) during the recovery phase, reveals intricate interactions between the viral infection and hepatic health. The liver function markers, including aspartate aminotransferase (AST), alanine aminotransferase (ALT), and albumin, exhibit notable variations that warrant careful consideration in the context of pre-existing metabolic conditions.

The elevated levels of AST and ALT, particularly in individuals recovering from COVID-19 with T2DM, suggest potential hepatic injury or stress during the post-infection period. These findings align with emerging evidence that indicates a high prevalence of liver abnormalities in COVID-19 patients, with reports of elevated liver enzymes and hepatocellular injury (Zhang et al., 2020). The liver, being a vital organ involved in various metabolic processes, may experience direct viral invasion or indirect damage mediated by the systemic inflammatory response induced by the virus.

Several potential mechanisms may contribute to the observed impact on liver function. Firstly, the direct cytopathic effect of the virus on hepatic cells is plausible. Studies have detected the presence of SARS-CoV-2 in liver tissues, suggesting the possibility of viral replication and damage to hepatocytes (Zhang et al., 2020). This viral tropism to the liver may contribute to the observed elevation in liver enzymes.

Secondly, the systemic inflammation triggered by COVID-19 may play a significant role in hepatic dysfunction. The heightened inflammatory response, characterized by increased levels of cytokines such as interleukin-6 (IL-6), can lead to hepatocellular damage and compromise liver function (Bellido & Pérez, 2021). The observed alterations in liver function markers may, in part, be a consequence of this inflammatory cascade.

Additionally, the compromised albumin levels in individuals recovering from COVID-19 with T2DM may signify impaired synthetic function of the liver. Albumin, a key protein synthesized by the liver, is essential for maintaining oncotic pressure and transporting various substances in the bloodstream. Reduced albumin levels may reflect liver dysfunction and impaired synthetic capacity, which can have implications for overall metabolic homeostasis.

Comparisons with existing studies reinforce the understanding that COVID-19 can exert a discernible impact on liver function. Reports of elevated liver enzymes and hepatic injury have been consistently documented in COVID-19 patients, with variations in the prevalence of liver abnormalities across different cohorts (Zhang et al., 2020; Bellido & Pérez, 2021). These findings collectively emphasize the need for heightened vigilance in monitoring liver health, especially in individuals with underlying metabolic conditions like T2DM, during the recovery phase from COVID-19.

The impact of COVID-19 on lipid metabolism, as reflected in the lipid profile of individuals with Type 2 Diabetes Mellitus (T2DM) during the recovery phase, unveils a complex interplay between the viral infection and lipid homeostasis. Lipid profile markers, including total cholesterol, triglycerides, high-density lipoprotein (HDL), and low-density lipoprotein (LDL), exhibit significant alterations, highlighting potential implications for cardiovascular health and metabolic balance.

The observed increase in total cholesterol and triglyceride levels in individuals recovering from COVID-19 with T2DM is noteworthy and aligns with emerging evidence suggesting dyslipidemia as a feature of COVID-19. Previous studies have reported lipid metabolism abnormalities in COVID-19 patients, including elevated total cholesterol and triglycerides, which may be linked to the systemic inflammatory response induced by the virus (Zhang et al., 2020; Huang et al., 2020). The inflammatory milieu associated with COVID-19 could stimulate lipolysis and alter lipid transport, leading to the observed changes in total cholesterol and triglyceride levels.

Conversely, the significant reduction in HDL levels in individuals recovering from COVID-19 with T2DM is consistent with reports of decreased HDL levels in COVID-19 patients. Reduced HDL levels have been proposed as a potential marker of disease severity, with studies suggesting a negative correlation between HDL levels and the severity of respiratory symptoms and inflammation in COVID-19 patients (Wei et al., 2020). The underlying mechanisms for the decrease in HDL are multifaceted and may involve impaired reverse cholesterol transport, increased HDL catabolism, and alterations in lipid metabolism induced by the viral infection.

Moreover, the observed changes in LDL levels, with a significant decrease in individuals recovering from COVID-19 with T2DM (GROUP 2), merit attention. Studies have reported variable effects of COVID-19 on LDL levels, with some indicating a decrease, while others report an increase or no significant change (Huang et al., 2020; Wei et al., 2020). The heterogeneity in findings across studies underscores the need for further investigation into the specific mechanisms by which COVID-19 influences LDL metabolism.

Comparisons with existing literature suggest that dyslipidemia is a common feature in COVID-19 patients, potentially influenced by the inflammatory response and the direct impact of the virus on lipid metabolism. The altered lipid profile observed in individuals recovering from COVID-19 with T2DM accentuates the need for comprehensive cardiovascular risk management in this population.

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