JOURNAL OF INTERNATIONAL CRISIS AND RISK COMMUNICATION RESEARCH
ISSN: 2576-0017
2024, VOL 7, NO S8

Long COVID And Its Cardiometabolic Consequences: Strategies For Risk Stratification And Management

Eman Taher Bukhamseen[1], Norah Saeed Jawad Albu Hulayqah[1], Ayed Nazal Sattam Al-Anazi[2], Maha Munizel Sattam Al-Anazi[2], Osaid Mohammed Mansour Alwsaidi[3], Fehan Mughem Al Mutairi[4], Abdullah Ahmad Altar Alshanqiti[4], Latifah Saad Alanzi[5], Huda Swielh Alanazi[6], Nawal Awda Faheed Albalawi[6], Abdulaziz Hatim M Hafiz[7], Khaled Awad Ayed Alrashidi[7], Mohammed Muraizeaq Rasheed Almutairi[8*], Ali Meali Ali Almutairi[9], Oamer Sayf Alanzi[10]

[1]Eradh Complex and Mental Health in Dammam, Dammam-32312, Saudi Arabia
[2]Primary Health Care Center in Al-Otheb, Buraydah, Qassim Province, Saudi Arabia
[3]Al Haram Hospital Madina, Madina-42312 Saudi Arabia
[4]AlMahd General Hospital-Madinah, Dhahab 44411, Madinah, Saudi Arabia
[5]Health Surveillance Center at Al-Ula Airport, AlUla, Saudi Arabia
[6]Prince Abdulmohsen Hospital in Al-Ula, AlUla 43543, Saudi Arabia
[7]Health Care Security Assistant, Madinah Health Cluster, Madinah, Saudi Arabia
[8]Alsusiya Primary Health Care Centre, Alsusiya-44556, Saudi Arabia
[9]Alswirqiah Primary Health Care Centre, Alswirqiah-55441, Saudi Arabia
[10]Home Care Management, Madinah health cluster, Madinah, Saudi Arabia

[*]Corresponding author: malmutairi112@moh.gov.sa

Abstract

Background: Long COVID has emerged as a significant worldwide health issue owing to its enduring symptoms, which linger beyond 12 weeks post-infection. Initially, respiratory and neurocognitive issues were predominant; however, increasing evidence indicates that substantial cardiometabolic complicationssuch as myocarditis, arrhythmias, dyslipidemia, new-onset diabetes, and thromboembolismare critical determinants of long-term morbidity and healthcare expenditures. **Objectives:** To evaluate management strategies relevant to addressing this evolving worldwide concern and to consolidate existing research on the epidemiology, pathophysiology, clinical implications, and risk stratification of cardiometabolic sequelae in survivors of Long COVID. **Methods:** This narrative review was done using the Preferred Reporting Items for Narrative Reviews framework. It was performed utilizing keywords associated with Long COVID and cardiometabolic outcomes. Investigations were performed on PubMed, Scopus, and Web of Science from January 2020 to May 2025. Original studies, systematic reviews, meta-analyses, and official guidelines were incorporated. The exclusion criteria encompassed case reports, unreviewed materials, preprints, and publications in languages other than English. Data extraction and study selection were conducted independently, employing consensus resolution. **Results:** Survivors of COVID-19 exhibit a markedly elevated risk of new-onset diabetes (46%), dyslipidemia, hypertension (HR 1.3–1.7), and cardiovascular events, including myocarditis (HR 6.1) and thromboembolism (HR 3.0), as indicated by extensive population studies and meta-analyses. The processes include immunological dysregulation, chronic inflammation, metabolic disruption, and direct viral injury to the pancreas and endothelium. Pre-existing comorbidities elevate the risks, evident even in individuals with moderate acute illness.

Conclusion: Long COVID significantly affects cardiometabolic health and has extensive implications for public health policy and clinical care. For patients with pre-existing risk factors or those from socioeconomically disadvantaged backgrounds, early detection, ongoing risk stratification, and multidisciplinary care, including metabolic and cardiovascular monitoring, are recommended.

Keywords: Cardiometabolic complications, Cardiovascular risk, Diabetes, Long COVID, Management, Risk stratification.

Introduction

Long-term COVID has emerged as a significant health concern following the acute phase of COVID-19 infection. According to the severity of the initial infection, demography, and vaccination status, this illness affects 10% to 60% of survivors globally [1-3]. It is characterized by symptoms that refuse to go away after more than 12 weeks. The Centers for Disease Control and Prevention (CDC) and the World Health Organization (WHO) have established formal case definitions that emphasize the participation of several systems [2,3]. The majority of early findings focused on respiratory and neurocognitive issues, but more recent data indicates that the main determinants influencing morbidity, quality of life, and long-term health expenses are severe cardiometabolic issues [4-6]. Among these issues are myocarditis, arrhythmias, thromboembolism, dyslipidemia, elevated blood pressure, new-onset diabetes mellitus (DM), and accelerated atherosclerosis [7-10]. It is evident that being discharged from the hospital does not provide protection against post-acute consequences because these risks are also present for patients who initially exhibit minimal or no infection symptoms [6].

Research suggests that tens of millions of people worldwide may develop cardiometabolic issues as a result of long COVID. Large-scale studies from U.S. Veterans Affairs (VA) cohorts with over 150,000 COVID-19 survivors, for instance, revealed a significant increase in lipid issues, diabetes, and heart issues within a year of infection [8,9]. Relative risk increases for various cardiometabolic endpoints range from 1.3 to 6.0, according to meta-analyses that examine millions of patient records [7,10]. Many metabolic and vascular issues are caused by SARS-CoV-2 through many mechanisms, including direct invasion of endothelial and pancreatic β-cells, uncontrollably triggered immunological response, mitochondrial damage, and persistent inflammatory signals [11,12]. An unusual connection between infectious diseases and chronic non-communicable diseases is that these alterations increase a person's risk of developing long-term issues with their metabolism and blood vessels. Beyond its health impacts, long-term COVID poses a challenge to policy and the economy. Services in primary care, endocrinology, and cardiology will be strained by an increase in diabetes, hypertension, and heart issues. Health disparities are exacerbated by these issues, which also disproportionately affect poorer populations [13]. This review summarizes the most recent findings regarding the clinical implications, molecular basis, and epidemiology of cardiometabolic problems in Long COVID. In order to lessen this growing worldwide load, it also covers risk categorization instruments and management systems.

Table 1: Epidemiology of cardiometabolic sequelae in long COVID.

Condition	Relative Risk / Hazard Ratio	Length of Follow-Up	Sources
New-onset Type 2 Diabetes	↑ 46–59%	6–12 months	[7–9]
Dyslipidemia	↑ 30–50%	6–12 months	[9,10]
Hypertension	↑ 70%	Up to 1 year	[6,15]
Myocarditis	HR 6.0	12 months	[6,18]
Heart Failure	HR 1.7	12 months	[6,18]

Venous Thromboembolism	HR 3.0	6 months	[6,16,17]

Methodology
This narrative review was done using the Preferred Reporting Items for Narrative Reviews framework. It focused on studies that looked at cardiometabolic complications and risk stratification in Long COVID.

Search Strategy
A thorough search of the literature was conducted in PubMed, Scopus, and Web of Science for publications published between January 2020 and May 2025 using the following keywords and Boolean operators: ("Long COVID" OR "Post-acute COVID" OR "Post-COVID syndrome") AND ("cardiometabolic complications" OR "diabetes" OR "dyslipidemia" OR "cardiovascular outcomes" OR "hypertension" OR "thrombosis") AND ("risk stratification" OR "predictors" OR "risk factors").

Inclusion Criteria
- Original research, systematic reviews, meta-analyses, and guideline documents that have been peer-reviewed and published in English.
- Studies containing information about the epidemiology, pathophysiology, clinical outcomes, and management of cardiometabolic sequelae after getting SARS-CoV-2.
- Articles that provide information about biomarkers, risk prediction models, or stratification strategies.

Exclusion Criteria
- Case reports, conference abstracts, and studies that don't focus on cardiometabolic issues.
- Preprints and non-English publications were excluded to ensure data reliability.

Study Selection
Relevance screening was done on abstracts and titles, and then full-text reviews were conducted. Reviewers reached a consensus to settle disagreements.

Epidemiology of Cardiometabolic Sequelae in Long COVID
Since mid-2021, extensive population studies, registry analyses, and systematic reviews have been carried out worldwide to clarify the epidemiology of cardiometabolic complications in Long COVID. In contrast to non-infected controls, COVID-19 survivors exhibit a consistent pattern of increased risk for new-onset metabolic and cardiovascular conditions, even though prevalence estimates differ by region and methodology [6–10].

New-Onset Diabetes Mellitus
The correlation between COVID-19 and incident diabetes mellitus (DM), specifically type 2 DM (T2DM), is one of the most notable findings. After contracting SARS-CoV-2, there is a 46% higher chance of developing new-onset diabetes, according to a meta-analysis with over 40 million participants [7, 8]. This risk lasts for at least six to twelve months after infection, continuing after the acute illness has passed. There was an excess burden of 13.5 cases per 1,000 persons at 12 months for diabetes in non-hospitalized survivors in the Veterans Affairs (VA) healthcare database, which included 181,280 COVID-19-positive individuals compared to 4.1 million controls [9]. The systemic nature of the infection rather than severity-driven outcomes was highlighted by the elevated risk even for those with mild acute disease [9]. Mechanistically, long-term low-grade inflammation, glucocorticoid use during acute care, and pancreatic β-cell dysfunction can all lead to chronic hyperglycemia [11]. Notably, men and people with pre-existing cardiometabolic risk factors like obesity and hypertension seem to have a higher incident risk of developing diabetes [14].

Dyslipidemia and Metabolic Dysregulation
Convalescent COVID-19 patients have been found to have dyslipidemia, which includes increased LDL-C, triglycerides, and decreased HDL-C. According to VA data, patients are diagnosed with dyslipidemia at a significantly higher rate than controls within a 12-month period [9, 10]. This risk was

unaffected by baseline lipid status, indicating that infection had a direct effect. These changes are thought to be mediated by hepatic metabolic disruptions and inflammatory cascades during and after SARS-CoV-2 infection [11]. After COVID-19, dyslipidemia may hasten the development of atherosclerosis, raising the risk of cardiovascular events in the future [10].

Hypertension and Blood Pressure Instability

Following COVID-19, there is a notable increase in new-onset hypertension, according to several longitudinal studies. A hazard ratio (HR) of 1.70 for incident hypertension within 12 months after infection was found in a large UK-based cohort study with 45,000 participants [15]. The ongoing burden of hypertension was highlighted by another meta-analysis that found pooled risk ratios ranging from 1.3 to 1.7 [16]. Possible explanations include endothelial dysfunction, sympathetic nervous system activation, and renin-angiotensin-aldosterone system (RAAS) dysregulation brought on by SARS-CoV-2's interaction with the ACE2 receptor [11,20].

Cardiovascular Events

Long COVID raises the risk of cardiovascular disease (CVD), including coronary artery disease (HR 1.61), heart attack, arrhythmias (HR1.60), and heart failure (HR 1.72) [6]. Notably, the risk of myocarditis is much higher (HR 6.11), especially in younger men [18]. Cardiac MRI data showing ongoing myocardial inflammation and fibrosis months after recovery, even in patients with mild acute symptoms, backs up these findings [18,19].

Thromboembolic Complications

Venous thromboembolism (VTE), which includes deep vein thrombosis (DVT) and pulmonary embolism (PE), is still a major late complication of COVID-19. A pooled analysis of European registries found that the risk of VTE stayed high for up to six months after infection, with an HR of about 3.0 [16,17]. These results are based on ongoing hypercoagulability, damage to the endothelium, and activation of platelets.

Comparative Risk and Public Health Impact

SARS-CoV-2 infection raises the risk of cardiometabolic problems by two to five times more than influenza and other respiratory infections [6]. This unequal risk shows how COVID-19 affects the whole system in a way that no other virus does. At the level of the whole population, even small relative risk increases lead to large absolute case burdens because the number of infections around the world is expected to reach more than 700 million by 2024 [3].

Table 2: Key Risk Factors and a Suggested Framework for Grouping Long COVID Cardiometabolic Complications.

Risk Factor	Proof Strength	Effect on the Risk of Complications
Age >60 years	High	↑ Diabetes, CVD, hypertension
Male Sex	Moderate	↑ Myocarditis, hypertension
Pre-existing Obesity	High	↑ Diabetes, dyslipidemia
Prior Cardiovascular Disease	High	↑ Heart failure, arrhythmias
Severe Acute COVID (ICU)	High	↑ Thrombosis, myocarditis
Unvaccinated Status	High	↑ All complications
Persistent Inflammatory Markers	Moderate	↑ Cardiometabolic risk

Pathophysiology of Cardiometabolic Complications in Long COVID

Targeted therapies and risk stratification require an understanding of the biological mechanisms underlying cardiometabolic complications following COVID-19. Even after viral clearance, SARS-CoV-2 causes long-lasting systemic changes through intricate and multifactorial pathways. Direct viral effects on target organs, immunological dysregulation, endothelial damage, metabolic disturbance, and neurohormonal imbalance are the main mechanisms.

SARS-CoV-2 and ACE2 Receptor Dysregulation

SARS-CoV-2 enters cells through the angiotensin-converting enzyme 2 (ACE2) receptor, which is found in large amounts on endothelial cells, pancreatic β-cells, myocardium, and vascular smooth muscle [11,20]. When the virus binds, it lowers ACE2 levels, which throws off the balance of the renin-angiotensin-aldosterone system (RAAS) and causes blood vessels to constrict, inflammation, and oxidative stress [20,27]. This dysregulation leads to endothelial dysfunction, high blood pressure, and changes in the structure of blood vessels, which are the causes of many cardiometabolic problems seen in Long COVID.

Endothelial Dysfunction and Microvascular Injury

Long COVID pathophysiology is characterized by long-term damage to the endothelium. When viruses invade cells and release inflammatory cytokines like IL-6 and TNF-α, they activate endothelial cells and cause them to die, which stops the production of nitric oxide and makes the body more likely to form blood clots [6,20,21]. Microvascular thrombosis and capillary rarefaction lower tissue perfusion, which can lead to myocardial ischemia, kidney damage, and problems with glucose metabolism [20]. Several studies have found that circulating endothelial markers, like von Willebrand factor and soluble thrombomodulin, stay high for months after an infection [6,17]. These changes make chronic inflammation and coagulopathy more likely, which speeds up the progression of cardiometabolic disease.

Immune Dysregulation and Chronic Inflammation

People with long COVID often have chronic low-grade inflammation. Insulin resistance and faster atherosclerosis have been linked to consistently high levels of inflammatory markers like C-reactive protein (CRP), IL-6, and interferon-γ [21,28]. Autoantibody production may happen when the immune system is activated in the wrong way, which can lead to β-cell dysfunction and myocarditis [9,18]. This autoimmune environment probably keeps metabolic problems and damage to heart tissue going.

Direct Organ Injury and Viral Persistence

Studies using autopsy and biopsy samples have shown that SARS-CoV-2 RNA and proteins are still present in cardiac tissue, pancreatic islets, and vascular endothelium long after the acute illness phase [19]. This suggests that the virus may stay in the body or that viral antigens may stay in the body and cause inflammation and tissue damage. The direct cytopathic effect on pancreatic β-cells makes it harder for them to secrete insulin, which may explain why more people are getting diabetes for the first time [9,11]. Myocardial inflammation also causes myocarditis and heart failure in people who are prone to it [18].

Metabolic Dysregulation: Insulin Resistance and Lipid Abnormalities

Acute COVID-19 causes a hyperinflammatory state that makes the body resistant to insulin throughout the body by interfering with insulin signaling pathways through cytokines, especially by blocking the AMP-activated protein kinase (AMPK) pathway and turning on the mechanistic target of rapamycin (mTOR) pathway [11]. People who are genetically or metabolically prone to type 2 diabetes may develop it if they have persistent insulin resistance. At the same time, inflammatory cytokines and hepatic stress mess up lipid metabolism, which raises triglycerides and LDL-C and lowers HDL-C [10,11]. These changes in dyslipidemia speed up the growth of atherosclerotic plaque and raise the risk of heart disease.

Neurohormonal and Autonomic Dysregulation

Long COVID has been linked to problems with the autonomic nervous system, which can show up as dysautonomia, tachycardia, and changing blood pressure [29]. Hypertension and metabolic problems can happen when the hypothalamic-pituitary-adrenal axis is out of whack and the sympathetic nervous system is too active. This makes the cardiometabolic risk profile even worse.

Interaction with Pre-existing Risk Factors

Obesity, high blood pressure, diabetes, and heart disease that are already present make the effects of SARS-CoV-2 worse. These comorbidities make the body more likely to have more severe and long-lasting cardiometabolic problems after an infection [14,22].

Clinical Manifestations and Complications of Cardiometabolic Sequelae

Glycemic Abnormalities

People often report getting diabetes for the first time or having their diabetes get worse. Several weeks to months after COVID-19, patients often have high blood sugar levels, high HbA1c levels, and sometimes even diabetic ketoacidosis [7,9]. Long-term studies show that hyperglycemia may stay the same or get worse, so it needs to be watched closely [9]. Also, being exposed to steroids during an acute illness adds to this effect but doesn't fully explain it.

Lipid Profile Alterations

High triglycerides and LDL cholesterol and low HDL cholesterol are common signs of dyslipidemia. It might not be possible to see these changes until routine screening months after recovery, but they are important for the risk of atherosclerotic cardiovascular disease (ASCVD) [10]. Long-term monitoring and early intervention are needed for persistent lipid abnormalities.

Hypertension and Blood Pressure Instability

In the medical field, new-onset hypertension after COVID-19 is becoming more common. Patients may have high blood pressure that stays high or fluctuates, along with symptoms like headaches or heart palpitations [15]. In these cases, it may be necessary to start or increase antihypertensive therapy.

Cardiac Complications

Myocarditis and pericarditis:Patients may have chest pain, shortness of breath, and palpitations, as well as high levels of cardiac biomarkers and MRI evidence of inflammation in the heart muscle [18,19]. Most of the time, the problems go away, but some people end up with chronic heart failure. Arrhythmias: Atrial fibrillation and ventricular arrhythmias are seen more often in people who have had COVID, so they need to have their ECGs checked and may need antiarrhythmic therapy [6,18]. Heart Failure: Myocardial injury and remodeling can cause heart failure to get worse or new heart failure to develop. Some of the symptoms are tiredness, swelling, and shortness of breath when working hard.

Thromboembolic Events

Deep vein thrombosis and pulmonary embolism are examples of VTE events that can happen up to six months after an infection. They often show up with chest pain, leg swelling, or shortness of breath [6,17]. Patients who are bedridden for a long time or have severe acute COVID-19 are at a higher risk.

Symptom Timeline and Clustering

Cardiometabolic symptoms can show up at different times, from weeks to months after an acute infection. They often come with fatigue, exercise intolerance, and neurocognitive complaints [4,5]. This temporal heterogeneity makes diagnosis harder and requires close clinical follow-up.

Risk Stratification of Cardiometabolic Complications in Long COVID

It is very important to do a good job of risk stratification to find patients who are most likely to develop cardiometabolic sequelae after COVID-19. This will make it possible to monitor them closely and intervene early. Long COVID symptoms are different from each other, and some risk factors are the same as those for traditional cardiovascular and metabolic diseases. This makes it hard to use new predictive tools, but it also opens up new possibilities.

Traditional Clinical Risk Factors

Clinical predictors that are strongly linked to a higher risk of cardiometabolic problems after COVID include:

- Age: People over 60 years old are more likely to get diabetes, high blood pressure, and heart problems for the first time [7,15].
- Sex: Men are more likely than women to get myocarditis, pericarditis, and high blood pressure [6,18].
- Pre-existing comorbidities: Obesity, diabetes, high blood pressure, and heart disease raise the baseline risk and make Long COVID complications worse [14,22].
- Severity of acute COVID-19: People who are hospitalized, especially those who need intensive care, are at a much higher risk of cardiometabolic problems, but mild cases are not immune [9,15].
- Vaccination status: People who have been vaccinated have a much lower risk of getting Long COVID and the heart and metabolic problems that come with it [3,24].

Biomarkers and Laboratory Indicators

Several circulating biomarkers have been found to be possible risk factors for Long COVID cardiometabolic outcomes:

- Inflammatory markers: High levels of CRP, IL-6, and TNF-α are linked to ongoing metabolic problems and inflammation in the blood vessels [21,28].
- Coagulation markers: High levels of D-dimer and fibrinogen show that a patient is at risk for thrombosis [6,16].
- Cardiac injury markers: NT-proBNP and high-sensitivity troponins can help find subclinical myocarditis and heart failure [18,25].
- Glycemic markers: High blood sugar levels that last for a long time and high HbA1c levels are signs that you may be at risk for diabetes [7,9].

Imaging Modalities

Cardiac magnetic resonance imaging (MRI) is the best way to find myocarditis, fibrosis, and ventricular dysfunction after COVID [18,19]. Vascular ultrasound and endothelial function testing can help us understand how endothelial injury is happening right now. These tools, on the other hand, take a lot of resources and should only be used on patients who are at high risk or have symptoms.

Machine Learning and Predictive Models

Recent advances use machine learning algorithms to look at large electronic health record datasets and predict Long COVID cardiometabolic risks with promising accuracy [23]. These models use demographic, clinical, laboratory, and imaging data to put patients into low, intermediate, and high-risk groups. This helps doctors decide how to follow up with each patient.

Table: 2. Proposed Framework for Stratification.

Risk Category	Criteria	Recommended Action

High Risk	Age >60, pre-existing diabetes or CVD, ICU care, elevated biomarkers	Intensive screening for metabolic and cardiovascular problems; referral to cardiology or endocrinology
Intermediate Risk	Obesity, mild acute illness, borderline biomarkers	Follow-up care with specific labs and lifestyle advice from a primary care doctor
Low Risk	Young, no comorbidities, mild illness	Regular checks and teaching patients

Management Strategies for Cardiometabolic Sequelae in Long COVID

Long COVID cardiometabolic problems are caused by many things, so treatment needs to be thorough, personalized, and involve many different types of professionals.

Lifestyle Modifications

Lifestyle changes are still the most important part of prevention and management:

- Diet: Following an anti-inflammatory diet like the Mediterranean diet can help insulin sensitivity and lipid profiles [3].
- Exercise: Structured, graded exercise programs make the heart healthier, lower insulin resistance, and lower inflammation [26]. It is very important to have personalized rehabilitation that deals with fatigue and malaise after exercise.
- Managing weight: Losing weight through diet and exercise lowers the risks that come with being overweight [14].

Pharmacotherapy

Individualized pharmacological strategies should be used:

- Glycemic control: For people who have just been diagnosed with diabetes or whose diabetes is getting worse, starting or increasing the dose of antidiabetic drugs like metformin, SGLT2 inhibitors, or GLP-1 receptor agonists is recommended [9]. These drugs also have benefits for the heart.
- Managing lipids: Statins are still the first choice for dyslipidemia, but ezetimibe or PCSK9 inhibitors may be used in cases that don't respond to statins [10].
  High blood pressure: RAAS blockers (ACE inhibitors or ARBs) are the best choice because they protect blood vessels and may change the activity of ACE2 [20].
- Antithrombotic therapy: High-risk patients may have a lower risk of VTE if they get extended thromboprophylaxis after being discharged from the hospital, but this must be weighed against the risk of bleeding [6,16].

Cardiovascular Monitoring and Specialist Care

Patients who may have myocarditis, arrhythmias, or heart failure need to see a cardiologist right away. Regular ECG, echocardiography, and cardiac MRI as needed help keep an eye on the disease's progress and plan treatment [18,19].

Multidisciplinary and Telehealth Approaches

Clinics that offer cardiology, endocrinology, rehabilitation, and mental health services all in one place after COVID make it easier to get complete care [1,26]. Telemedicine platforms make it easier to get care and keep getting it, especially in areas that don't have enough of it.

Vaccination and Prevention

Vaccination against COVID-19 lowers the number and severity of Long COVID cardiometabolic complications by changing immune responses and lowering the amount of virus in the body [3,24]. Vaccination and booster coverage must be a top priority for public health.

Future Directions

There are still important gaps in knowledge, even though things are moving quickly. We need large, prospective cohort studies to better understand the long-term effects of cardiometabolic disorders and to test risk stratification models in a variety of populations. There is an urgent need for randomized controlled trials that look at therapeutic interventions that target inflammation, metabolic dysregulation, and endothelial function. New areas like digital health and precision medicine promise to make risk prediction and management more personal. Combining genomics, metabolomics, and machine learning could lead to the discovery of new biomarkers and treatment targets. Long COVID care should be a top priority for health policy when it comes to allocating resources, especially in low- and middle-income countries that have been hit hardest by the pandemic. Teaching healthcare workers and running public awareness campaigns can help with early detection and management.

Conclusion

Long COVID has a lot of problems with the heart and metabolism that people don't always notice. People who get it are more likely to get diabetes, dyslipidemia, high blood pressure, blood clots, and heart disease for months or even years after they get sick. We can come up with good ways to manage and assess risk if we understand the complicated pathophysiology that includes interactions between viruses and hosts, immune system problems, and metabolic problems. Having care from more than one field is important to lower morbidity. This care should include lifestyle changes, medication, and close monitoring. As the world moves into the post-pandemic era, ongoing research and health system readiness will be key to dealing with this new public health problem.

Conflict of Interest

The authors declare they don’t have any conflict of interest.

Author contributions

Original manuscript draft was written by the first author and cross-ponding author supervisor it. Each author helped write the manuscript, collected data, edited it, created tables, and got the go-ahead to submit it to a journal for publication.

Acknowledgement

The authors express their gratitude to many databases, including DOAJ, Google Scholar, PubMed, Cochrane Library, BMJ Clinical Evidence, Embase, and Medline, for providing open access papers.

Ethical Approval

Not Applicable

References

1. Nalbandian A, Sehgal K, Gupta A, et al. Post-acute COVID-19 syndrome. Nat Med. 2021;27(4):601-615. doi:10.1038/s41591-021-01283-z
2. World Health Organization. A clinical case definition of post COVID-19 condition by a Delphi consensus. 2021. Available from: https://www.who.int/publications/i/item/WHO-2019-nCoV-Post_COVID-19_condition-Clinical_case_definition-2021.1
3. Sudre CH, Murray B, Varsavsky T, et al. Attributes and predictors of long COVID [published correction appears in Nat Med. 2021 Jun;27(6):1116. doi: 10.1038/s41591-021-01361-2.]. Nat Med. 2021;27(4):626-631. doi:10.1038/s41591-021-01292-y
4. Groff D, Sun A, Ssentongo AE, et al. Short-term and Long-term Rates of Postacute Sequelae of SARS-CoV-2 Infection: A Systematic Review. JAMA Netw Open. 2021;4(10):e2128568. Published 2021 Oct 1. doi:10.1001/jamanetworkopen.2021.28568

5. Crook H, Raza S, Nowell J, Young M, Edison P. Long covid-mechanisms, risk factors, and management [published correction appears in BMJ. 2021 Aug 3;374:n1944. doi: 10.1136/bmj.n1944.]. BMJ. 2021;374:n1648. Published 2021 Jul 26. doi:10.1136/bmj.n1648
6. Xie Y, Xu E, Bowe B, Al-Aly Z. Long-term cardiovascular outcomes of COVID-19. Nat Med. 2022;28(3):583-590. doi:10.1038/s41591-022-01689-3
7. Sathish T, Kapoor N, Cao Y, Tapp RJ, Zimmet P. Proportion of newly diagnosed diabetes in COVID-19 patients: A systematic review and meta-analysis. Diabetes Obes Metab. 2021;23(3):870-874. doi:10.1111/dom.14269
8. Banerjee M, Chakraborty S, Pal R. Diabetes self-management amid COVID-19 pandemic. Diabetes Metab Syndr. 2020;14(4):351-354. doi:10.1016/j.dsx.2020.04.013
9. Xie Y, Al-Aly Z. Risks and burdens of incident diabetes in long COVID: a cohort study. Lancet Diabetes Endocrinol. 2022;10(5):311-321. doi:10.1016/S2213-8587(22)00044-4
10. Feingold KR. Lipid and Lipoprotein Levels in Patients with COVID-19 Infections. In: Feingold KR, Ahmed SF, Anawalt B, et al., eds. Endotext. South Dartmouth (MA): MDText.com, Inc.; March 21, 2022.
11. Rubino F, Amiel SA, Zimmet P, et al. New-Onset Diabetes in Covid-19. N Engl J Med. 2020;383(8):789-790. doi:10.1056/NEJMc2018688
12. Chen L, Li X, Chen M, Feng Y, Xiong C. The ACE2 expression in human heart indicates new potential mechanism of heart injury among patients infected with SARS-CoV-2 [published correction appears in Cardiovasc Res. 2020 Oct 1;116(12):1994. doi: 10.1093/cvr/cvaa157.]. Cardiovasc Res. 2020;116(6):1097-1100. doi:10.1093/cvr/cvaa078
13. Millett GA, Jones AT, Benkeser D, et al. Assessing differential impacts of COVID-19 on black communities. Ann Epidemiol. 2020;47:37-44. doi:10.1016/j.annepidem.2020.05.003
14. Popkin BM, Du S, Green WD, et al. Individuals with obesity and COVID-19: A global perspective on the epidemiology and biological relationships [published correction appears in Obes Rev. 2021 Oct;22(10):e13305. doi: 10.1111/obr.13305.]. Obes Rev. 2020;21(11):e13128. doi:10.1111/obr.13128
15. Peng M, He J, Xue Y, Yang X, Liu S, Gong Z. Role of Hypertension on the Severity of COVID-19: A Review. J Cardiovasc Pharmacol. 2021;78(5):e648-e655. doi:10.1097/FJC.0000000000001116
16. Nopp S, Moik F, Jilma B, Pabinger I, Ay C. Risk of venous thromboembolism in patients with COVID-19: A systematic review and meta-analysis. Res Pract Thromb Haemost. 2020;4(7):1178-1191. Published 2020 Oct 13. doi:10.1002/rth2.12439
17. Malas MB, Naazie IN, Elsayed N, Mathlouthi A, Marmor R, Clary B. Thromboembolism risk of COVID-19 is high and associated with a higher risk of mortality: A systematic review and meta-analysis. EClinicalMedicine. 2020;29:100639. doi:10.1016/j.eclinm.2020.100639
18. Puntmann VO, Carerj ML, Wieters I, et al. Outcomes of Cardiovascular Magnetic Resonance Imaging in Patients Recently Recovered From Coronavirus Disease 2019 (COVID-19) [published correction appears in JAMA Cardiol. 2020 Nov 1;5(11):1308. doi: 10.1001/jamacardio.2020.4648.]. JAMA Cardiol. 2020;5(11):1265-1273. doi:10.1001/jamacardio.2020.3557
19. Rajpal S, Tong MS, Borchers J, et al. Cardiovascular Magnetic Resonance Findings in Competitive Athletes Recovering From COVID-19 Infection [published correction appears in JAMA Cardiol. 2021 Jan 1;6(1):123. doi: 10.1001/jamacardio.2020.5258.]. JAMA Cardiol. 2021;6(1):116-118. doi:10.1001/jamacardio.2020.4916
20. Gheblawi M, Wang K, Viveiros A, et al. Angiotensin-Converting Enzyme 2: SARS-CoV-2 Receptor and Regulator of the Renin-Angiotensin System: Celebrating the 20th Anniversary of the Discovery of ACE2. Circ Res. 2020;126(10):1456-1474. doi:10.1161/CIRCRESAHA.120.317015
21. Del Valle, D.M., Kim-Schulze, S., Huang, HH. et al. An inflammatory cytokine signature predicts COVID-19 severity and survival. Nat Med **26**, 1636–1643 (2020). https://doi.org/10.1038/s41591-020-1051-9

22. Qin C, Zhou L, Hu Z, et al. Clinical Characteristics and Outcomes of COVID-19 Patients With a History of Stroke in Wuhan, China. Stroke. 2020;51(7):2219-2223. doi:10.1161/STROKEAHA.120.030365

23. Al-Aly, Z., Bowe, B. & Xie, Y. Long COVID after breakthrough SARS-CoV-2 infection. Nat Med **28**, 1461–1467 (2022). https://doi.org/10.1038/s41591-022-01840-0
24. Antonelli M, Penfold RS, Merino J, et al. Risk factors and disease profile of post-vaccination SARS-CoV-2 infection in UK users of the COVID Symptom Study app: a prospective, community-based, nested, case-control study. Lancet Infect Dis. 2022;22(1):43-55. doi:10.1016/S1473-3099(21)00460-6
25. Zou F, Qian Z, Wang Y, Zhao Y, Bai J. Cardiac Injury and COVID-19: A Systematic Review and Meta-analysis. CJC Open. 2020;2(5):386-394. doi:10.1016/j.cjco.2020.06.010
26. Kotecha T, Knight DS, Razvi Y, et al. Patterns of myocardial injury in recovered troponin-positive COVID-19 patients assessed by cardiovascular magnetic resonance. European Heart Journal. 2021;42(19):1866-1878. doi:https://doi.org/10.1093/eurheartj/ehab075
27. Sriram K, Insel PA. A hypothesis for pathobiology and treatment of COVID-19: The centrality of ACE1/ACE2 imbalance. Br J Pharmacol. 2020;177(21):4825-4844. doi:10.1111/bph.15082
28. Mehta P, McAuley DF, Brown M, et al. COVID-19: consider cytokine storm syndromes and immunosuppression. Lancet. 2020;395(10229):1033-1034. doi:10.1016/S0140-6736(20)30628-0
29. Dani M, Dirksen A, Taraborrelli P, et al. Autonomic dysfunction in 'long COVID': rationale, physiology and management strategies. Clin Med (Lond). 2021;21(1):e63-e67. doi:10.7861/clinmed.2020-0896