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COVID- 19 AND MYOCARDIAL INFRACTION: UNVEILING THE COMPLEX RELATIONSHIP

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ABSTRACT

A complex relationship between viral infection and cardiovascular health, particularly in connection to myocardial infarction (MI) or heart attacks, has been highlighted by the COVID-19 pandemic. The respiratory tract, not the heart, is the target of the seven human corona viruses that are now known to exist. Severe Acute Respiratory Syndrome Coronavirus 2 (SARS-CoV-2) is recent corona virus which also affect the heart. It can cause myocarditis, which is an inflammation of the heart, cell necrosis, arrhythmias, and either acute or chronic heart failure, which is caused by muscle dysfunction. These problems have happened even in cases with minor symptoms and in those who did not experience any symptoms. Among many other places, we thoroughly searched PubMed and Google Scholar. In order to clarify the complex link between COVID-19 and MI, this review looks at recent research, covering topics such as underlying pathophysiological mechanisms, clinical updates on cardiovascular manifestation of covid-19, adverse effects of COVID-19 in relation to MI, diagnostic challenges and strategies in covid-19 with MI and therapeutic approaches. After mRNA COVID-19 vaccinations, a no of systemic and local adverse effects have been seen. Myocarditis, pericarditis, and myocardial infarction have been linked with these vaccinations. Acute coronary syndrome treatment has been impacted at every stage by the COVID-19 pandemic, which has placed an unparalleled amount of strain on healthcare systems. COVID-19 not only increasing the risk of heart problems but also has a direct effect on its tissue through hypercoagulability, systemic inflammation and also endothelial dysfunction. It is imperative to comprehend these pathways in order to promptly identify and treat MI in COVID-19 patients. The study also examines therapeutic issues and identifies information gaps, promoting ongoing research to improve clinical outcomes in this susceptible population. Unfortunately, a wide range of clinical indications of multisystem dysfunction, including pulmonary embolism, deep vein thrombosis, acute myocardial infarction, depression, anxiety, myalgia, dyspnea, and fatigue, may still be reported by recovered COVID-19 survivors.

Keywords: COVID-19, MI, SARS-Cov-2, Cardiovascular Complications, Myocarditis, Pericarditis, Systematic Review.

I. INTRODUCTION

The complex links between viral infections and cardiovascular illnesses has been seen by SARS-CoV-2 new coronavirus which causes COVID-19 pandemic. Research investigations and emerging clinical reports have brought attention to a troubling correlation with COVID-19 and a higher risk of myocardial infarction (MI), also referred to as heart attacks. There are several facets to this association, including intricate interactions between systemic inflammatory responses, pre-existing cardiovascular risk factors, and viral pathogenesis. Although COVID-19 is most known for its respiratory symptoms, it has gradually been apparent how it affects cardiovascular health. It can appear as mild myocardial damage, acute coronary syndromes, fulminant myocarditis, or any combination of these conditions. It is imperative to comprehend the fundamental mechanisms that give rise to these cardiovascular problems in order to enhance therapeutic therapy and ameliorate patient outcomes. This review aims to conclusively explore the current understanding of the relationship between COVID-19 and myocardial infarction. It synthesizes available epidemiological data, discusses proposed pathophysiological mechanisms linking viral infection to cardiovascular events, and examines clinical implications for diagnosis, management, and future research directions. By elucidating these complexities, this review seeks to provide a foundation for enhancing clinical awareness, guiding therapeutic strategies, and fostering continued investigation into mitigating the cardiovascular impact of COVID-19. Myocardial infarction (MI), arrhythmias, cardiac arrests, heart failure and coagulation abnormality are some of the manifestation of cardiovascular system in COVID-19 which ranging from 7.2% up to 33% [1]. The most frequent presentations were fever (38.2%) and cardiac arrest (96.1%). There were increases in CK-MB, and troponin in 100%, and 99.5%, of the individuals, respectively. An abnormal ST segment was the most common

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ECG finding. All individuals with myocarditis were found to have abnormal cardiac magnetic resonance imaging, the gold standard for detecting the condition [21].

II. MECHANISM: COVID-19 AND MI

The globe is currently experiencing a terrifying worldwide pandemic as a result of the COVID-19 virus and SARS-CoV-2 virus [3]. The mechanism linking COVID-19 and myocardial infarction (MI), or heart attack, involves several interconnected pathways that contribute to cardiovascular complications in infected individuals.

Globally, acute coronary syndromes are a major contributor to morbidity and mortality. A number of interrelated pathways are involved in the process that links COVID-19 to myocardial infarction (MI), or heart attacks, and these pathways can lead to cardiovascular problems in infected patients. Numerous factors, such as elevated systemic and coronary inflammatory activity, predominate prothrombotic conditions, increased biomechanical stress on coronary arteries, changes in coronary arterial tone, disrupted hemodynamic homoeostasis, and modified myocardial metabolic balance, are responsible for these events. Acute infections have been shown in experiments to facilitate the development of acute coronary syndromes, and clinical evidence is strongly in favor of acute infections playing a role in cardiac events. [2].

In individuals with COVID-19, cardiac involvement should be considered a probable late manifestation of the viral respiratory infection. Some research suggests that the mechanism of cardiac involvement in COVID-19 patients involves direct injury to myocardial cells mediated by angiotensin-converting enzyme 2 (ACE2) receptors; however, other research suggests that systemic inflammation may also be involved, leading to indirect myocyte injury. Effective triage, vigilant supervision, and avoiding certain medications with cardiovascular toxicity are crucial for managing circulatory system involvement in COVID-19 patients. Cardiovascular system involvement in COVID-19 individuals is common, erratic, and incapacitating. As a result, it needs our focus and careful supervision [1].

The main mechanisms are:

- SARS-CoV-2, the virus that causes COVID-19, penetrates human cells through the ACE2 receptor, which is expressed in cardiovascular organs such as the heart and blood vessels. This is known as the "direct viral effect" on cardiovascular tissues. Through the disruption of cellular function and induction of inflammation, viral entrance into these cells can directly cause cardiac damage.[4]
- Systemic Inflammation and Cytokine Storm: COVID-19 frequently triggers a cytokine storm, an augmented immune response marked by the production of chemokines and pro-inflammatory cytokines. The pathophysiology of MI can be significantly influenced by endothelial dysfunction, plaque instability, and thrombosis, all of which can be exacerbated by this systemic inflammation.[5]
- Thrombosis and Endothelial Dysfunction: The inflammation brought on by the virus can harm the endothelial cells that line blood arteries, which can result in endothelial dysfunction. Due to the pro-thrombotic condition this dysfunction fosters, there is a higher chance of arterial thrombosis and consequent myocardial ischemia [6].
- Cardiovascular Risk Factors Pre-existing: People with pre-existing cardiovascular risk factors, such as hypertension, diabetes, obesity, and coronary artery disease, are disproportionately affected by COVID-19. These diseases independently raise the risk of MI in addition to making one more susceptible to a severe COVID-19 infection [7].
- Hemodynamic instability and mismatch between cardiac oxygen supply and demand: Severe respiratory disease linked to COVID-19 can cause hypoxia and systemic hypotension, which can impair the oxygen flow to the heart. MI can be triggered by this imbalance between the supply and demand of oxygen in the heart, particularly in individuals who already have coronary artery disease [8].
- Drug-Induced Effects: Certain antiviral drugs and corticosteroids, which are used to treat COVID-19, may have direct or indirect cardiovascular effects that worsen pre-existing cardiovascular problems or cause myocardial injury [9].

Understanding these mechanisms is crucial for guiding clinical management strategies, including early detection and intervention to mitigate the risk of myocardial infarction in COVID-19 patients. Further research

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is needed to unravel the precise interplay between COVID-19 infection and cardiovascular complications, thereby improving outcomes for affected individuals.

III. CARDIOVASCULAR MANIFESTATION OF COVID-19: CLINICAL UPDATES

COVID-19 can affect the cardiovascular system in several ways, leading to various clinical features:

- Myocarditis: Inflammation of the heart muscle can occur, causing symptoms like chest pain, shortness of breath, and arrhythmias (irregular heart rhythms) [7][20].
- Arrhythmias: COVID-19 can disrupt the normal electrical activity of the heart, leading to palpitations, dizziness, or fainting [10].
- Acute Coronary Syndromes: This includes conditions like myocardial infarction (heart attack), where there is a sudden blockage of blood flow to the heart due to clot formation or other mechanisms.
- Heart Failure: COVID-19 can exacerbate pre-existing heart failure or lead to new-onset heart failure, characterized by symptoms such as shortness of breath, fatigue, and swelling of the legs.
- Thromboembolism: Formation of blood clots can occur in COVID-19 patients, increasing the risk of conditions like pulmonary embolism (clot in the lungs) or stroke.
- Pericarditis: Inflammation of the pericardium (the sac around the heart) can cause chest pain that worsens with deep breathing or lying down.
- Hypertension: COVID-19 infection can worsen blood pressure control in hypertensive patients or lead to new-onset hypertension.
- Endothelial Dysfunction: The virus can directly affect the endothelium (inner lining of blood vessels), leading to vascular inflammation and dysfunction.
- Kawasaki-like Disease in Children: Some children infected with COVID-19 may develop a multisystem inflammatory syndrome (MIS-C) that can involve the cardiovascular system, resembling Kawasaki disease.

These cardiovascular manifestations underscore the importance of monitoring heart health in COVID-19 patients, especially those with pre-existing cardiovascular conditions. Early recognition and management of these complications are crucial in improving outcomes for patients with COVID-19.

IV. ADVERSE EFFECTS OF COVID-19 IN RELATION TO MI

Polycystic The COVID-19 pandemic has profoundly impacted cardiovascular health, particularly in its association with myocardial infarction (MI). Evidence suggests that COVID-19 not only increases the incidence of MI but also exacerbates its severity and complicates management strategies.

- Direct Myocardial Injury: SARS-CoV-2 may directly infect myocardial cells through the ACE2 receptor, leading to myocarditis and acute myocardial injury[11], increased cardiovascular risk and mortality[7], systemic inflammation and cytokine storm: severe COVID-19 infection triggers a cytokine storm, contributing to endothelial dysfunction, thrombosis, and plaque instability, all of which increase the risk of MI [5], exacerbation of Pre-existing Cardiovascular Conditions: Patients with underlying cardiovascular risk factors are particularly vulnerable to severe COVID-19 outcomes, including MI [8], thrombotic complications: COVID-19-induced hypercoagulability increases the risk of arterial thrombosis and subsequent MI [6], delayed recognition and treatment: Pandemic-related healthcare disruptions have led to delays in recognizing MI symptoms and accessing timely interventions, impacting patient outcomes[12].

Understanding these adverse effects is crucial for optimizing clinical management strategies and improving outcomes for COVID-19 patients at risk of MI. Further research is essential to delineate the precise mechanisms and long-term consequences of COVID-19 on cardiovascular health.

V. DIAGNOSTIC CHALLENGES AND STRATEGIES IN COVID-19 WITH MI

Clinical Evaluation: Given the overlap of symptoms between COVID-19 and MI (such as chest pain and shortness of breath), a thorough clinical evaluation is crucial. Physicians assess the patient's history, risk factors for cardiovascular disease, and the presence of typical or atypical symptoms.

Electrocardiogram (ECG): An ECG is a fundamental diagnostic tool for detecting MI. It helps identify characteristic changes such as ST-segment elevation (STEMI) or ST-segment depression (NSTEMI). In COVID-

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19 patients, ECG findings may be complicated by electrolyte imbalances, medications, or systemic inflammation.

Cardiac Biomarkers: Measurement of cardiac biomarkers such as troponin is essential for confirming myocardial injury. Elevated troponin levels indicate myocardial damage and are crucial in diagnosing MI in COVID-19 patients, even in the absence of typical symptoms [13].

Imaging Studies: Echocardiography: Used to assess cardiac structure and function, including regional wall motion abnormalities indicative of MI and coronary angiography: In severe cases or when indicated, coronary angiography may be performed to visualize coronary artery anatomy and identify obstructive lesions.

Multimodal Approach: Given the complexities in diagnosing MI in COVID-19 patients, a multimodal diagnostic approach integrating clinical evaluation, ECG findings, cardiac biomarkers, and imaging studies is often necessary for accurate diagnosis and management.

These diagnostic strategies emphasize the importance of a comprehensive approach tailored to the unique clinical and pathophysiological features of COVID-19 to ensure prompt identification and management of myocardial infarction.

VI. COVID-19 AND MI: THERAPEUTIC INSIGHTS

Managing COVID-19 in patients who also present with myocardial infarction (MI) requires a careful and integrated therapeutic approach. Firstly initial assessment and stabilization, using oxygen therapy to maintain adequate oxygen saturation and assess for signs shock or heart failure [14]. Continuous cardiac monitoring, ECG and frequent assessment of vital signs are crucial. Pharmacological Therapy: antiplatelet therapy: Initiate aspirin and consider P2Y12 inhibitors (e.g., clopidogrel, ticagrelor) for dual antiplatelet therapy (DAPT) unless contraindicated and anticoagulation: start heparin or low-molecular-weight heparin (LMWH) to prevent further thrombotic events, balancing with bleeding risks. Use opioids or other analgesics cautiously due to respiratory depression risk in COVID-19 patients for pain management. Consider early. Percutaneous Coronary Intervention (PCI) if feasible and indicated by clinical presentation, with adherence to infection control measures. Thrombolysis: Use fibrinolytic therapy in eligible patients when PCI is not available or delayed, weighing risks and benefits carefully.

COVID-19-Specific Therapies:

Antiviral Therapy: Consider remdesivir for hospitalized patients with severe COVID-19, based on severity and timing of symptoms [15].

Anti-inflammatory Therapy: Use corticosteroids (e.g., dexamethasone) in severe cases requiring supplemental oxygen or mechanical ventilation, based on inflammatory markers and clinical status [16].

Supportive Care:

Respiratory Support: Provide supplemental oxygen; escalate to non-invasive ventilation or mechanical ventilation as needed.

Fluid Management: Balance fluid administration carefully to avoid volume overload, particularly in patients with compromised cardiac function [17].

Cardioprotective Measures:

Beta-blockers: Initiate beta-blockers unless contraindicated, to reduce myocardial oxygen demand and prevent arrhythmias.

ACE Inhibitors/ARBs: Continue or initiate ACE inhibitors or ARBs as indicated for heart failure or hypertension management, balancing risks and benefits [18].

Follow-up and Rehabilitation:

Cardiac Rehabilitation: Initiate post-acute care to optimize cardiovascular health and improve functional status.

Telemedicine: Use telemedicine for follow-up visits and monitoring, particularly important for ongoing COVID-19 recovery and cardiac rehabilitation [19].

Multidisciplinary Approach:

Collaboration: Involve cardiologists, infectious disease specialists, intensivists, and allied health professionals for comprehensive management and decision-making.

This integrated therapeutic approach aims to address both the acute cardiovascular complications of MI and the systemic effects of COVID-19, optimizing outcomes through careful coordination and management of therapies.

VII. CONCLUSION

The intersection of COVID-19 and myocardial infarction presents significant clinical challenges and complexities. COVID-19 not only predisposes patients to cardiovascular complications such as MI through inflammatory pathways and thrombotic mechanisms but also complicates the management of acute coronary syndromes due to systemic effects and potential drug interactions. The therapeutic approach requires a nuanced balance between antiviral, antiplatelet, anticoagulant, and supportive therapies tailored to each patient's clinical presentation and severity of illness. Moreover, the integration of multidisciplinary care involving cardiologists, infectious disease specialists, intensivists, and allied health professionals is essential for optimizing outcomes and minimizing mortality. Future research should continue to elucidate the pathophysiological links between COVID-19 and MI and explore innovative strategies for prevention, early detection, and management of these intertwined conditions."

This conclusion summarizes the key points discussed in the review article, highlighting the challenges, therapeutic strategies, multidisciplinary approach, and the need for ongoing research in managing COVID-19 patients with MI.

VIII. REFERENCES

[1] Kwenandar F, Japar KV, Damay V, Hariyanto TI, Tanaka M, Lugito NPH, Kurniawan A. Coronavirus disease 2019 and cardiovascular system: A narrative review. Int J Cardiol Heart Vasc. 2020 Jun 3;29:100557. doi: 10.1016/j.ijcha.2020.100557. PMID: 32550259; PMCID: PMC7266760.

[2] Corrales-Medina VF, Madjid M, Musher DM. Role of acute infection in triggering acute coronary syndromes. Lancet Infect Dis. 2010 Feb;10(2):83-92.
doi: 10.1016/S1473-3099(09)70331-7. PMID: 20113977.

[3] Mahenthiran AK, Mahenthiran AK, Mahenthiran J. Cardiovascular system and COVID-19: manifestations and therapeutics. Rev Cardiovasc Med. 2020 Sep 30;21(3):399-409.
doi: 10.31083/j.rcm.2020.03.124. PMID: 33070544.

[4] 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. Cardiovasc Res. 2020 May 1;116(6):1097-1100. doi: 10.1093/cvr/cvaa078. Erratum in: Cardiovasc Res. 2020 Oct 1;116(12):1994. doi: 10.1093/cvr/cvaa157. PMID: 32227090; PMCID: PMC7184507.

[5] Mehta P, McAuley DF, Brown M, Sanchez E, Tattersall RS, Manson JJ; HLH Across Speciality Collaboration, UK. COVID-19: consider cytokine storm syndromes and immunosuppression. Lancet. 2020 Mar 28;395(10229):1033-1034. doi: 10.1016/S0140-6736(20)30628-0. Epub 2020 Mar 16. PMID: 32192578; PMCID: PMC7270045.

[6] Libby P, Lüscher T. COVID-19 is, in the end, an endothelial disease. Eur Heart J. 2020 Sep 1;41(32):3038-3044. doi: 10.1093/eurheartj/ehaa623. PMID: 32882706; PMCID: PMC7470753.

[7] Guo T, Fan Y, Chen M, Wu X, Zhang L, He T, Wang H, Wan J, Wang X, Lu Z. Cardiovascular Implications of Fatal Outcomes of Patients With Coronavirus Disease 2019 (COVID-19). JAMA Cardiol. 2020 Jul 1;5(7):811-818. doi: 10.1001/jamacardio.2020.1017. Erratum in: JAMA Cardiol. 2020 Jul 1;5(7):848. doi: 10.1001/jamacardio.2020.1722. PMID: 32219356; PMCID: PMC7101506.

[8] Bangalore S, Sharma A, Slotwiner A, Yatskar L, Harari R, Shah B, Ibrahim H, Friedman GH, Thompson C, Alviar CL, Chadow HL, Fishman GI, Reynolds HR, Keller N, Hochman JS. ST-Segment Elevation in Patients with Covid-19 - A Case Series. N Engl J Med. 2020 Jun 18;382(25):2478-2480. doi: 10.1056/NEJMc2009020. Epub 2020 Apr 17. PMID: 32302081; PMCID: PMC7182015.

[9] Alhazzani, W., Møller, M.H., Arabi, Y.M. et al. Surviving Sepsis Campaign: guidelines on the management of critically ill adults with Coronavirus Disease 2019 (COVID-19). Intensive Care Med **46**, 854–887 (2020). https://doi.org/10.1007/s00134-020-06022-5

[10] Kochav SM, Coromilas E, Nalbandian A, Ranard LS, Gupta A, Chung MK, Gopinathannair R, Biviano AB, Garan H, Wan EY. Cardiac Arrhythmias in COVID-19 Infection. Circ Arrhythm Electrophysiol. 2020 Jun;13(6):e008719. doi: 10.1161/CIRCEP.120.008719. Epub 2020 May 20. PMID: 32434385; PMCID: PMC7299099.

[11] Inciardi RM, Lupi L, Zaccone G, Italia L, Raffo M, Tomasoni D, Cani DS, Cerini M, Farina D, Gavazzi E, Maroldi R, Adamo M, Ammirati E, Sinagra G, Lombardi CM, Metra M. Cardiac Involvement in a Patient With Coronavirus Disease 2019 (COVID-19). JAMA Cardiol. 2020 Jul 1;5(7):819-824. doi: 10.1001/jamacardio.2020.1096. PMID: 32219357; PMCID: PMC7364333.

[12] Garcia S, Albaghdadi MS, Meraj PM, Schmidt C, Garberich R, Jaffer FA, Dixon S, Rade JJ, Tannenbaum M, Chambers J, Huang PP, Henry TD. Reduction in ST-Segment Elevation Cardiac Catheterization Laboratory Activations in the United States During COVID-19 Pandemic. J Am Coll Cardiol. 2020 Jun 9;75(22):2871-2872. doi: 10.1016/j.jacc.2020.04.011. Epub 2020 Apr 10. PMID: 32283124; PMCID: PMC7151384.

[13] Sandoval Y, Januzzi JL Jr, Jaffe AS. Cardiac Troponin for Assessment of Myocardial Injury in COVID-19: JACC Review Topic of the Week. J Am Coll Cardiol. 2020 Sep 8;76(10):1244-1258. doi: 10.1016/j.jacc.2020.06.068. Epub 2020 Jul 8. PMID: 32652195; PMCID: PMC7833921.

[14] https://www.who.int/initiatives/oxygen-access-scale-up

[15] Ali K, Azher T, Baqi M, Binnie A, Borgia S, Carrier FM, Cavayas YA, Chagnon N, Cheng MP, Conly J, Costiniuk C, Daley P, Daneman N, Douglas J, Downey C, Duan E, Duceppe E, Durand M, English S, Farjou G, Fera E, Fontela P, Fowler R, Fralick M, Geagea A, Grant J, Harrison LB, Havey T, Hoang H, Kelly LE, Keynan Y, Khwaja K, Klein G, Klein M, Kolan C, Kronfli N, Lamontagne F, Lau R, Fralick M, Lee TC, Lee N, Lim R, Longo S, Lostun A, MacIntyre E, Malhamé I, Mangof K, McGuinty M, Mergler S, Munan MP, Murthy S, O'Neil C, Ovakim D, Papenburg J, Parhar K, Parvathy SN, Patel C, Perez-Patrigeon S, Pinto R, Rajakumaran S, Rishu A, Roba-Oshin M, Rushton M, Saleem M, Salvadori M, Scherr K, Schwartz K, Semret M, Silverman M, Singh A, Sligl W, Smith S, Somayaji R, Tan DHS, Tobin S, Todd M, Tran TV, Tremblay A, Tsang J, Turgeon A, Vakil E, Weatherald J, Yansouni C, Zarychanski R; Canadian Treatments for COVID-19 (CATCO); Association of Medical Microbiology and Infectious Disease Canada (AMMI) Clinical Research Network and the Canadian Critical Care Trials Group. Remdesivir for the treatment of patients in hospital with COVID-19 in Canada: a randomized controlled trial. CMAJ. 2022 Feb 22;194(7):E242-E251. doi: 10.1503/cmaj.211698. Epub 2022 Jan 19. PMID: 35045989; PMCID: PMC8863204.

[16] National Institutes of Health. COVID-19 Treatment Guidelines. Antiviral Therapy in Adults with COVID-19. Available from: https://www.covid19treatmentguidelines.nih.gov/antiviral-therapy/

[17] Alhazzani W, Møller MH, Arabi YM, Loeb M, Gong MN, Fan E, Oczkowski S, Levy MM, Derde L, Dzierba A, Du B, Aboodi M, Wunsch H, Cecconi M, Koh Y, Chertow DS, Maitland K, Alshamsi F, Belley-Cote E, Greco M, Laundy M, Morgan JS, Kesecioglu J, McGeer A, Mermel L, Mammen MJ, Alexander PE, Arrington A, Centofanti JE, Citerio G, Baw B, Memish ZA, Hammond N, Hayden FG, Evans L, Rhodes A. Surviving Sepsis Campaign: guidelines on the management of critically ill adults with Coronavirus Disease 2019 (COVID-19). Intensive Care Med. 2020 May;46(5):854-887. doi: 10.1007/s00134-020-06022-5. Epub 2020 Mar 28. PMID: 32222812; PMCID: PMC7101866. Alame AJ, Karatasakis A, Karacsonyi J, Danek BA, Resendes E, Martinez Parachini JR, Kalsaria P, Roesle M, Rangan BV, Sorajja P, Jneid H, Banerjee S, Brilakis ES. Comparison of the American College of Cardiology/American Heart Association and the European Society of Cardiology guidelines for the management of patients with non-ST-segment elevation acute coronary syndromes. Coron Artery Dis. 2017 Jun;28(4):294-300. doi: 10.1097/MCA.0000000000000472. PMID: 28306587.

[18] Alame AJ, Karatasakis A, Karacsonyi J, Danek BA, Resendes E, Martinez Parachini JR, Kalsaria P, Roesle M, Rangan BV, Sorajja P, Jneid H, Banerjee S, Brilakis ES. Comparison of the American College of Cardiology/American Heart Association and the European Society of Cardiology guidelines for the management of patients with non-ST-segment elevation acute coronary syndromes. Coron Artery Dis. 2017 Jun;28(4):294-300. doi: 10.1097/MCA.0000000000000472. PMID: 28306587.

[19] Anderson L, Oldridge N, Thompson DR, Zwisler AD, Rees K, Martin N, Taylor RS. Exercise-Based Cardiac Rehabilitation for Coronary Heart Disease: Cochrane Systematic Review and Meta-Analysis. J Am Coll Cardiol. 2016 Jan 5;67(1):1-12. doi: 10.1016/j.jacc.2015.10.044. PMID: 26764059.

[20] Yasmin F, Najeeb H, Naeem U, Moeed A, Atif AR, Asghar MS, Nimri N, Saleem M, Bandyopadhyay D, Krittanawong C, Fadelallah Eljack MM, Tahir MJ, Waqar F. Adverse events following COVID-19 mRNA vaccines: A systematic review of cardiovascular complication, thrombosis, and thrombocytopenia. Immun Inflamm Dis. 2023 Mar;11(3):e807. doi: 10.1002/iid3.807. PMID: 36988252; PMCID: PMC10022421.

[21] Fazlollahi A, Zahmatyar M, Noori M, Nejadghaderi SA, Sullman MJM, Shekarriz-Foumani R, Kolahi AA, Singh K, Safiri S. Cardiac complications following mRNA COVID-19 vaccines: A systematic review of case reports and case series. Rev Med Virol. 2022 Jul;32(4):e2318. doi: 10.1002/rmv.2318. Epub 2021 Dec 17. PMID: 34921468.