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Case Report

Late onset ST-elevation myocardial infarction (STEMI) in patient with COVID-19: A case report from Nepal

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ABSTRACT

Introduction: Although pulmonary consequences are less common in COVID-19 than cardiac issues, it is critical to understand the cause of probable cardiac complications and put the patient on constant watch, especially if they have risk factors such as diabetes mellitus.

Case presentation: Here, we report a case of 82-years old male with ST-segment elevated myocardial infarction (STEMI) that was developed as a complication of COVID-19.

Discussion: COVID-19 is now known to cause cardiovascular issues such as myocardial damage, heart failure, arrhythmia, and venous thromboembolism. With the involvement of COVID-19, the prevalence of cardiovascular manifestation has increased. The precise processes of extrapulmonary and systemic manifestations following COVID-19 are unknown. There is an elevated risk of cardiovascular harm, notably myocardial infraction followed by acute infection.

Conclusion: It is essential to understand the mechanism of potential cardiac complications and to keep the patient on close watch, especially if the patient has risk factors such as diabetes mellitus.

1. Introduction

Coronavirus disease (COVID-19) is an infectious disease caused by severe acute respiratory syndrome coronavirus-2 (SARS-CoV-2) and causes symptoms that range from mild respiratory symptoms to severe respiratory and cardiovascular problems. It is believed that COVID-19 is transmitted via aerosol [1]. Cardiac complication includes acute myocardial infarction (MI), myocarditis, heart failure, arterial and venous thromboembolic events [2]. The mechanism by which SARS-CoV-2 causes cardiovascular complications is that it binds to the angiotensin-converting enzyme-2 receptor (ACE-2) present on the endothelial surface of alveolar and cardiac cells [3]. Thrombus formation in a COVID-19 patient may be a result of abnormal platelet activation, impaired fibrinolysis, and endothelial dysfunction [4]. Here, we report a case of 82-years old male with ST-segment elevated myocardial infarction (STEMI) that was developed as a complication of COVID-19. This case has been reported in line with SCARE 2020 criteria [5].

2. Case presentation

An 82-year-old male presented to our center with chief complaints of chest pain, chest heaviness, and sweating for 2 hours. Chest pain was radiating towards jaw. He had no limb swelling, shortness of breath, palpitation, and dizziness. He had type 2 diabetes mellitus for two years. He was tested positive for COVID-19 five months back which was confirmed by reverse transcriptase polymerase chain reaction (RT-PCR). He did not smoke but consumed alcohol occasionally.

He was in distress due to pain and was ill looking, not well oriented to time, place, and person. On general physical examination, he had no pallor, icterus, clubbing, cyanosis, or edema. His blood pressure was 130/70 mm of Hg, heart rate 62 beats/minute, oxygen saturation (SpO2) 91% in room air, respiratory rate 20 breaths per minute, and axillary temperature was 36.5 °C. He had vesicular breath sound. First and second heart sounds were heard on auscultation.

His laboratory investigation revealed hemoglobin 14.7 g %, total leucocyte count 12450 cells/mm^3, neutrophils 77%, lymphocytes 13%,

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and platelets 253000 cells/mm^3. The cardiac markers; creatinine kinase-MB (CK-MB) was 64 units/liter, and Troponin I was 0.346 ng/ml. His random blood sugar (RBS) was 569 mg/dl, HbA_1c 11.5%, sodium 128mmol/L, potassium 5.1 mmol/L, urea 16 mg/dl, and creatinine 1.26 mg/dl. Liver function test showed total protein 7.8 g/dl, total bilirubin 0.8 mg/dl, direct bilirubin 0.2 mg/dl, alanine aminotransferase (SGPT) 45 U/L, and aspartate aminotransferase (SGOT) 41 U/L. The thyroid function test showed free T_3 3.38 pg/ml, free T_4 1.78 ng/dl, and TSH 0.17 uIU/ml. The HDL and LDL level in the blood was 1.8mmol/l and 3.2 mmol/l respectively. There was trace amount of albumin, high amount (++++) of sugar, and plenty of pus cells per high power field in urine. The immunochromatography test for HBsAg, anti HCV ab, and HIV 1 and 2 ab were non-reactive. Electrocardiogram (ECG) showed ST elevation in multiple leads viz anterior leads (V2–V6) (Fig. 1). His echocardiogram findings showed decrease in left ventricular ejection fraction (35%). Coronary angiography revealed double vessel disease (left anterior descending artery and left circumflex artery). Based on these findings diagnosis of anterior STEMI was made.

Lab parameters	Values
Hemoglobin	14.7 g %
Total leucocyte count	12450 cells/mm^3
Neutrophils	77%
Lymphocytes	13%
Platelets	253000 cells/mm^3
Creatinine kinase-MB (CK-MB)	64 units/liter
Troponin I	0.346 ng/ml
Random blood sugar (RBS)	569 mg/dl
HbA_1c	11.5%
Sodium	128mmol/L
Potassium	5.1 mmol/L
Urea	16 mg/dl
Creatinine	1.26 mg/dl
Total protein	7.8 g/dl
Total bilirubin	0.8 mg/dl
Direct bilirubin	0.2 mg/dl
Alanine aminotransferase (SGPT)	45 U/L
Aspartate aminotransferase (SGOT)	41 U/L
Free T_3	3.38 pg/ml
Free T_4	1.78 ng/dl
TSH	0.17 uIU/ml
HDL	1.8mmol/l
LDL	3.2 mmol/l

Following this, he underwent percutaneous transluminal coronary angioplasty (PTCA) of m-LAD. He was given IV cefazolin (1 gm), IV verapamil (2.5 mg), and Heparin (10000 IU) before the procedure. Drug eluting stents were used. Following the CAG, ECG was normal. He was in oral medications with aspirin (300 mg OD), clopidogrel (75mg BD), rosuvastatin (20mg OD), flucloxacillin (500mg QID), linagliptin (5 mg OD), and empagliflozin (10mg OD).

3. Discussion

There have been case reports of patients developing various cardiac manifestations such as myocardial infarction, myocarditis, and so on during COVID-19 [6–8]. However, in the long run, we don't know the effects of COVID-19 on the heart. It should be highlighted that the long-term effects of COVID-19 should be investigated. The long-term effect on various organ systems following COVID-19 infection should be investigated. In our patient with diabetes mellitus-II and a recent COVID-19, the manifestation of STEMI might be a late complication due to prior COVID-19. Diabetes is one of the risk factors for coronary heart disease which has role in development of STEMI [9]. One risk factor has helped other, and the cumulative effect has resulted in development of STEMI which cannot be ignored.

Infection with SARS-CoV-2 predominately causes respiratory illness ranging from mild disease to severe disease and death, while some people infected with the virus do not develop symptoms. According to current evidence, the main mechanism of transmission of the SARS-CoV-2 virus is via aerosols [10]. A susceptible person may inhale particles, which enter the lungs and infiltrate pulmonary cells, resulting in respiratory failure, pneumonia, or ARDS. However, it is now clear that COVID-19 causes cardiovascular problems such as myocardial damage, heart failure, arrhythmia, and venous thromboembolism. The frequency of cardiovascular manifestation has increased with the involvement of COVID-19 [11]. The exact mechanisms of extra-pulmonary and systemic manifestation following COVID-19 are very poorly understood. As per studies, there is an increased risk of cardiovascular injury, specifically myocardial infarction followed by acute infection [12–14].

The exact mechanism by which SARS-CoV-2 causes cardiovascular injury is unknown. However, several hypotheses about the pathophysiology of cardiovascular complications in COVID-19 patients have been proposed. The potential mechanisms by which SARS-CoV-2 enter cardiac cells is through receptor-mediated endocytosis. Spike protein of SARS-CoV-2 binds with the angiotensin-converting enzyme-2 (ACE-2) receptor. The ACE-2 receptor is present in the abundant amount of cardiac tissue [15]. In the severe form COVID-19 causes systemic inflammation triggering cytokine storms that led to multiorgan failure, including the cardiovascular system [16]. A condition of hypoxemia is created during respiratory failure which leads to mismatch in demand and supply ratio in cardiac cells, thus leading to acute myocardial injury. Another possible mechanism by which acute myocardial injury can cause is the embolism of microthrombi. SARS-CoV-2 is known to cause a

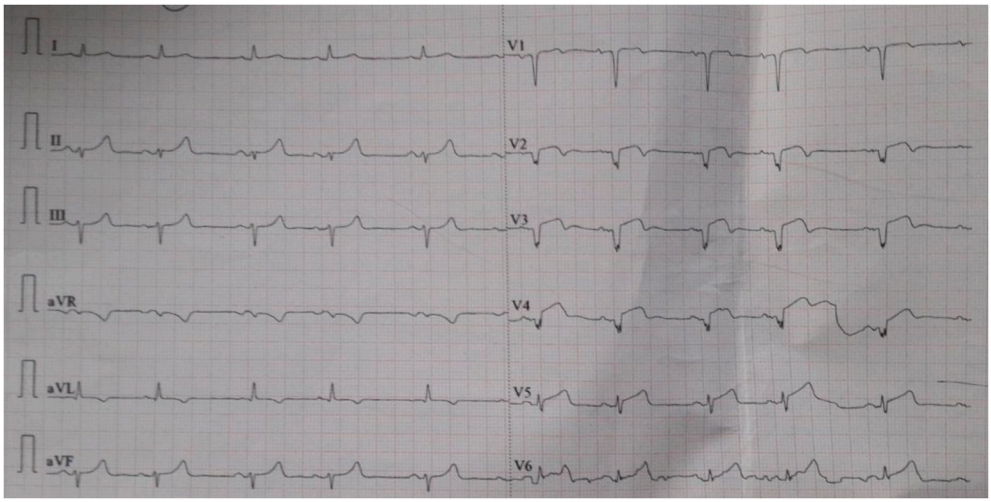

Fig. 1. ECG showing STEMI in a patient with COVID-19.

prothrombotic state which then results in the formation of microthrombi. The formed microthrombi then can embolize and result in acute ischemia [17]. In our case, this could be a process through which slowly developed microthrombi embolized and induce ischemia and ultimately lead to myocardial infarction.

In a recent prospective cohort study, Fardman et al. revealed that patients presenting with STEMI have a longer overall ischemia time, which translates into a more severe disease condition upon hospital admission and a higher likelihood of in-hospital ill effects [18]. There is high morbidity and mortality rate in patients with cardiac complications after COVID-19 [18]. STEMI in itself is a severe cardiac manifestation, when coupled with the COVID-19 leading to STEMI it becomes a matter of concern. Microthrombi formation, cytokine storm, and hypoxic injury are thought to be pathogenesis of acute myocardial infarction in COVID-19 patients.

4. Conclusion

Although pulmonary complications are frequent in comparison to cardiac complications in COVID-19, it is important to understand the mechanism of potential cardiac complications and keep the patient on a close follow-up, especially for the patients who have risk factors like diabetes mellitus.

Ethical approval

None.

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No funding was received for the study.

Authors' contributions

SS and PM wrote the original manuscript, reviewed, and edited the original manuscript. RC reviewed and edited the original manuscript. SS, PM, RC, LR, AS, SKS, AJ, RG, AK, and SA reviewed the manuscript and was in charge of case.

Trial registry number

1. Name of the registry: None
2. Unique Identifying number or registration ID: None
3. Hyperlink to your specific registration (must be publicly accessible and will be checked):

Guarantor

Sangam Shah.

Data availability statement

All the required data are in manuscript itself.

Consent

Written informed consent was obtained from the patient for publication of this case report and accompanying images. A copy of the written consent is available for review by the Editor-in-Chief of this journal on request.

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Declaration of competing interest

Authors have no conflict of interest to declare.

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References

[1] Priyanka, O.P. Choudhary, I. Singh, G. Patra, Aerosol transmission of SARS-CoV-2: the unresolved paradox, Travel Med. Infect. Dis. 37 (2020), https://doi.org/10.1016/J.TMAID.2020.101869.

[2] B. Long, W.J. Brady, A. Koyfman, M. Gottlieb, Cardiovascular complications in COVID-19, Am. J. Emerg. Med. 38 (2020) 1504–1507, https://doi.org/10.1016/j.ajem.2020.04.048.

[3] Inciardi: Cardiac Involvement in a Patient with Coronavir… - Google Scholar, (n.d.).

[4] S.X. Gu, T. Tyagi, K. Jain, V.W. Gu, S.H. Lee, J.M. Hwa, J.M. Kwan, D.S. Krause, A. I. Lee, S. Halene, K.A. Martin, H.J. Chun, J. Hwa, Thrombocytopathy and endotheliopathy: crucial contributors to COVID-19 thromboinflammation, Nat. Rev. Cardiol. 18 (2021) 1, https://doi.org/10.1038/S41569-020-00469-1.

[5] R.A. Agha, T. Franchi, C. Sohrabi, G. Mathew, A. Kerwan, A. Thoma, A.J. Beamish, A. Noureldin, A. Rao, B. Vasudevan, B. Challacombe, B. Perakath, B. Kirshtein, B. Ekser, C.S. Pramesh, D.M. Laskin, D. Machado-Aranda, D. Miguel, D. Pagano, F. H. Millham, G. Roy, H. Kadioglu, I.J. Nixon, I. Mukhejree, J.A. McCaul, J. Chi-Yong Ngu, J. Albrecht, J.G. Rivas, K. Raveendran, L. Derbyshire, M.H. Ather, M. A. Thorat, M. Valmasoni, M. Bashashati, M. Chalkoo, N.Z. Teo, N. Raison, O. J. Muensterer, P.J. Bradley, P. Goel, P.S. Pai, R.Y. Afifi, R.D. Rosin, R. Coppola, R. Klappenbach, R. Wynn, R.L. De Wilde, S. Surani, S. Giordano, S. Massarut, S. G. Raja, S. Basu, S.A. Enam, T.G. Manning, T. Cross, V.K. Karanth, V. Kasivisvanathan, Z. Mei, The SCARE 2020 guideline: updating consensus surgical CAse REport (SCARE) guidelines, Int. J. Surg. 84 (2020) 226–230, https://doi.org/10.1016/J.IJSU.2020.10.034.

[6] H.N. Kim, J.H. Lee, H.S. Park, D.H. Yang, S.Y. Jang, M.H. Bae, Y. Cho, S.C. Chae, Y. H. Lee, A case of COVID-19 with acute myocardial infarction and cardiogenic shock, J. Kor. Med. Sci. 35 (2020) 1–6, https://doi.org/10.3346/JKMS.2020.35.E258.

[7] D. Tedeschi, A. Rizzi, S. Biscaglia, C. Tumscitz, Acute myocardial infarction and large coronary thrombosis in a patient with COVID-19, Catheter. Cardiovasc. Interv. 97 (2021) 272–277, https://doi.org/10.1002/ccd.29179.

[8] A. Mansoor, D. Chang, R. Mitra, Rhythm, conduction, and ST elevation with COVID-19: myocarditis or myocardial infarction? Hear. Case Rep. 6 (2020) 671–675, https://doi.org/10.1016/j.hrcr.2020.08.001.

[9] F. M, Diabetes and myocardial infarction, Baillieres. Best Pract. Res. Clin. Endocrinol. Metab. 13 (1999) 331–343, https://doi.org/10.1053/BEEM.1999.0024.

[10] S. Tang, Y. Mao, R.M. Jones, Q. Tan, J.S. Ji, N. Li, J. Shen, Y. Lv, L. Pan, P. Ding, X. Wang, Y. Wang, C.R. MacIntyre, X. Shi, Aerosol transmission of SARS-CoV-2? Evidence, prevention and control, Environ. Int. 144 (2020) 106039, https://doi.org/10.1016/J.ENVINT.2020.106039.

[11] F.G.P. Welt, P.B. Shah, H.D. Aronow, A.E. Bortnick, T.D. Henry, M.W. Sherwood, M.N. Young, L.J. Davidson, S. Kadavath, E. Mahmud, A.J. Kirtane, Catheterization laboratory considerations during the coronavirus (COVID-19) pandemic: from the ACC's interventional council and SCAI, J. Am. Coll. Cardiol. 75 (2020) 2372–2375, https://doi.org/10.1016/J.JACC.2020.03.021.

[12] V.F. Corrales-Medina, M. Madjid, D.M. Musher, Role of acute infection in triggering acute coronary syndromes, Lancet Infect. Dis. 10 (2010) 83–92, https://doi.org/10.1016/S1473-3099(09)70331-7.

[13] L. Smeeth, S.L. Thomas, A.J. Hall, R. Hubbard, P. Farrington, P. Vallance, Risk of Myocardial Infarction and Stroke after Acute Infection or Vaccination, 2009, pp. 2611–2618, https://doi.org/10.1056/NEJMOA041747.

[14] C. Warren-Gash, A.C. Hayward, H. Hemingway, S. Denaxas, S.L. Thomas, A. D. Timmis, H. Whitaker, L. Smeeth, Influenza infection and risk of acute myocardial infarction in England and Wales: a CALIBER self-controlled case series study, J. Infect. Dis. 206 (2012) 1652–1659, https://doi.org/10.1093/INFDIS/JIS597.

[15] Y. Chen, Q. Liu, D. Guo, Emerging coronaviruses: genome structure, replication, and pathogenesis, J. Med. Virol. 92 (2020) 418–423, https://doi.org/10.1002/JMV.25681.

[16] C. Huang, Y. Wang, X. Li, L. Ren, J. Zhao, Y. Hu, L. Zhang, G. Fan, J. Xu, X. Gu, Z. Cheng, T. Yu, J. Xia, Y. Wei, W. Wu, X. Xie, W. Yin, H. Li, M. Liu, Y. Xiao, H. Gao, L. Guo, J. Xie, G. Wang, R. Jiang, Z. Gao, Q. Jin, J. Wang, B. Cao, Clinical features of patients infected with 2019 novel coronavirus in Wuhan, China, Lancet 395 (2020) 497–506, https://doi.org/10.1016/S0140-6736(20)30183-5.

[17] D. Pellegrini, R. Kawakami, G. Guagliumi, A. Sakamoto, K. Kawai, A. Gianatti, A. Nasr, R. Kutys, L. Guo, A. Cornelissen, L. Faggi, M. Mori, Y. Sato, I. Pescetelli, M. Brivio, M. Romero, R. Virmani, A.V. Finn, Microthrombi as a major cause of cardiac injury in COVID-19: a pathologic study, Circulation (2021) 1031–1042, https://doi.org/10.1161/CIRCULATIONAHA.120.051828.

[18] A.I. Fardman, D. Zahger, K. Orvin, D.I. Oren, N. Kofman, J. Mohsen, O. TsafrirID, E. Asher, R. Rubinshtein, J. Jamal, R. Efraim, M. Halabi, Y. Shacham, L. Henri FortisID, T. Cohen, R. Klempfner, A. Segev, R. Beigel, S. Matetzky, Acute

Myocardial Infarction in the Covid-19 Era: Incidence, Clinical Characteristics and In-Hospital Outcomes-A Multicenter Registry, 2021, https://doi.org/10.1371/journal.pone.0253524.