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Type 2 myocardial infarction in COVID19: World's first case series

Gaurav Jagtap[1], **Dhruv Talwar**[1]✉, **Sunil Kumar**[2], **Sourya Acharya**[3], **Iftekhar Ansari**[1], **Meenal Rajput**[4]

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Author Affiliation:
[1]Post Graduate Resident, Department of Medicine, Jawaharlal Nehru medical college, Datta Meghe Institute of Medical Sciences (Deemed to be University), Wardha, Maharashtra, India
[2]Professor, Department of Medicine, Jawaharlal Nehru medical college, Datta Meghe Institute of Medical Sciences (Deemed to be University), Wardha, Maharashtra, India
[3]Professor and Head of Department, Department of Medicine, Jawaharlal Nehru medical college, Datta Meghe Institute of Medical Sciences (Deemed to be University), Wardha, Maharashtra, India.
[4]Resident Medical Officer, Shri Shankaraacharya Instituite of Medical Science, Bhilai, Chhattisgarh, India

✉**Corresponding author**
Post Graduate Resident, Department of Medicine, Jawaharlal Nehru medical college, Datta Meghe Institute of Medical Sciences (Deemed to be University), Wardha, Maharashtra, India
Email: dhruv.talwar2395@gmail.com

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ABSTRACT

Type 2 Myocardial Infarction is characterized as a myocardial infarction which is caused by factors other than Coronary Artery Disease (CAD). The causative factor for a secondary myocardial infarction is a disbalance between oxygen supply and its demand. There have been cases where there was 90% blockage in the coronaries but still myocardial infarction was classified as type 2 as the causative factor was not Coronary artery disease but the disbalance between oxygen demand and supply. Thus there is a diagnostic challenge with considerable overlap in Type 2 and Type 1 Myocardial Infarction. COVID19 has brought a tremendous load on healthcare ever since it's identification in the late 2019. There is increased demand of Oxygen due to hypoxia caused by severe pneumonia leading to a disbalance between oxygen supply and demand causing a type 2 myocardial infarction, though the cases of Type 1 myocardial infarction due to COVID19 are well documented this is the first case series showing Type 2 myocardial infarction in the world to our knowledge.

Keywords: COVID19, Pneumonia, Type 2 Myocardial Infarction

1. INTRODUCTION

In December 2019, Wuhan witnessed the first case of COVID19 which led to a series of unfortunate spread leading to a deadly pandemic. It has been a difficult disease to manage due its unforeseeable outcomes and presentations that have been different in different cases. Presentation of COVID19 can range from Severe Pneumonia, Myocardial Infarction to a Stroke. Recent reports have also shown Psychosis and Hiccups as presenting features of COVID19. Although, Type 1 Myocardial Infarction is a well documented complication of COVID19 owing to the coagulopathy caused by COVID 19, Type 2 Myocardial Infarction have not yet been documented anywhere in the world. With the evidence of Severe Hypoxia caused due to COVID19, the absence of Type 2 Myocardial Infarction is hard to believe. Type 2 Myocardial Infarction is myocardial necrosis seen due to diseases other than Coronary Artery Disease. It is caused due to imbalance of oxygen demand and supply of the myocytes (Thygesen et al., 2012). Common causes of increased demand of oxygen are anemia, hypotension, endothelial dysfunction and hypoxemia while common

causes of decreased oxygen supply can be sespsis, arrhythmia or tachycardia (Alpert et al., 2009). Sepsis is more often associated with Type 2 myocardial infarction presenting with ST Elevation Myocardial Infarction rather than Non ST elevation myocardial infarction. This can be explained by the fact that Non ST elevation Myocardial infarction is caused due to incomplete occlusion of Coronary Arteries (Pierport et al., 2009). Diagnosing Type 2 Myocardial Infarction can be challenging as there is a significant overlap between type 1 and type 2 myocardial infarction.

Here we report a case series of 5 patients who had severe COVID19 pneumonia and had severe hypoxia which resulted in a Type 2 Myocardial Infarction diagnosed after normal coronary angiography.

2. CASE SERIES

Case 1

50 year old Male presented to the outpatient department with the chief complaint of High Grade Fever with breathlessness since 4 days. There was no history of diabetes mellitus, hypertension or coronary artery disease and other chronic medical conditions. There was no history of ciggerate smoking or malignancy in the past. Patient had no history of Sore Throat, Cough, chest pain or Nasal Congestion.

On General Physical Examination, patient was febrile with temperature of 101 degree Fahrenheit, pulse was 77 beats per minute, regular in rhythm and volume, Blood pressure was 126/80mm hg in right arm supine position, respiratory rate was 34 breaths per minute and oxygen saturation was 88% on room air. Systemic Examination revealed bilateral bronchial breath sounds. An HRCT of Chest was carried out which showed multiple ill defined patchy ground glass opacities with consolidation and septal thickening in bilateral lung fields suggestive of viral pneumonia with a CT Severity Score of 22/25 and CORAD 5 (figure 1). Nasopharyngeal Swab was sent for Reverse Transcription Polymerase Chain Reaction (RTPCR) which turned out to be Positive for COVID19. Patient was managed on Remdesavir Antibiotics, low molecular weight heparin, dexamethasone, Ivermectin, high flow oxygen and other supportive measures. Patient was taken on non invasive ventilation on day 4 of admission in view of falling saturation. During the course of hospital stay on day 6 of admission patient developed severe chest pain and ECG was done showing changes of acute myocardial infarction (Figure 3). The patient was taken for immediate coronary angiography which revealed normal coronaries (Figure 2). Hence a diagnosis of Type 2 Myocardial Infarction was made.

Patient was supported further with non invasive ventilation to treat hypoxemia and was weaned off and taken on high flow oxygen which was slowly tapered and stopped on day 20 of admission. Patient was discharged on day 22 of admission in stable condition and is doing well on follow up.

Case 2

73 year old Male presented to the outpatient department with the chief complaint of chest pain which was left sided compressing type with breathlessness since 16 hours. There was history of diabetes mellitus for which he was on oral hypoglycemics. There was no history of hypertension or coronary artery disease and other chronic medical conditions. There was no history of ciggerate smoking or alcohol abuse or malignancy in the past. Patient had no history of fever, Sore Throat, Cough, chest pain or Nasal Congestion.

On General Physical Examination, patient was afebrile, pulse was 127 beats per minute, regular in rhythm and volume, Blood pressure was 136/84mm hg in right arm supine position, respiratory rate was 38 breaths per minute and oxygen saturation was 80% on room air. Systemic Examination revealed bilateral bronchial breath sounds, heart sounds were normal, patient was conscious and oriented and abdominal examination was normal with no organomegaly or tenderness. Patient was admitted in ICU and started on high flow oxygen, antibiotics, steroids with other supportive measures

Nasopharyngeal Swab was sent for Reverse Transcription Polymerase Chain Reaction (RTPCR) which turned out to be positive for COVID19. An HRCT of Chest was carried out which showed multiple ill defined patchy ground glass opacities with consolidation and septal thickening in bilateral lung fields suggestive of viral pneumonia with a CT Severity Score of 20/25 and CORAD 6 (figure 1). Patient was managed on Remdesavir, Antibiotics, low molecular weight heparin, dexamethasone, Ivermectin, and other supportive measures. Patient was taken on non invasive ventilation in view of falling saturation. ECG was done showing changes of acute myocardial infarction (Figure 4). The patient was taken for immediate coronary angiography which revealed normal coronaries (Figure 2). Hence a diagnosis of Type 2 Myocardial Infarction was made. Patient was supported further with non invasive ventilation to treat hypoxemia but did not maintain oxygen saturation and was hence intubated on day 7 of admission and ultimately succumbed on day 10.

Case 3

39 year old Male presented to the outpatient department with the chief complaint of cough with breathlessness since 2 days. There was no history of hypertension or coronary artery disease and other chronic medical conditions. There was no history of ciggerate smoking or alcohol abuse or malignancy in the past. Patient had no history of fever, Sore Throat, Cough, chest pain or Nasal Congestion. On General Physical Examination, patient was afebrile; pulse was 98 beats per minute, regular in rhythm and volume. Blood pressure was 128/78mm hg in right arm supine position, respiratory rate was 38 breaths per minute and oxygen saturation was 84% on room air. Systemic Examination revealed bilateral bronchial breath sounds rest of the systemic examination was unremarkable. Patient was admitted in ICU and started on high flow oxygen, antibiotics, steroids with other supportive measures Nasopharyngeal Swab was sent for Reverse Transcription Polymerase Chain Reaction (RTPCR) which turned out to be positive for COVID19. An HRCT of Chest was carried out which showed multiple ill defined patchy ground glass opacities with consolidation and septal thickening in bilateral lung fields suggestive of viral pneumonia with a CT Severity Score of 21/25 and CORAD 6 (figure 1). Patient was managed on Remdesavir, Antibiotics, low molecular weight heparin, dexamethasone, Ivermectin, and other supportive measures. Patient was taken on non invasive ventilation in view of falling saturation. During the course of hospital stay patient was not maintaining oxygen saturation and developed severe chest pain on 5th day of admission. ECG was done showing changes of acute myocardial infarction (Figure 5) .CKMB and Troponin I were raised. The patient was taken for immediate coronary angiography which revealed normal coronaries (Figure 2). Hence a diagnosis of Type 2 Myocardial Infarction was made. Hypoxemia was treated and patient was shifted to high flow oxygen and then ultimately discharged on 2 liter of oxygen support.

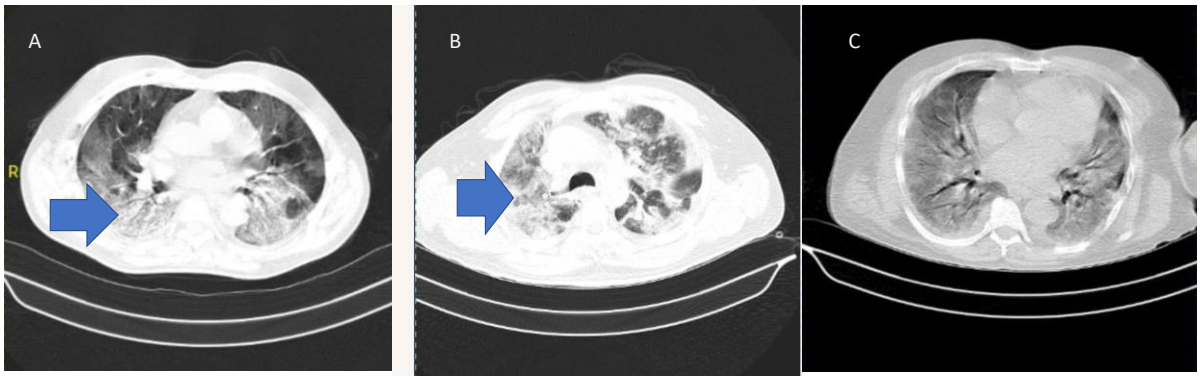
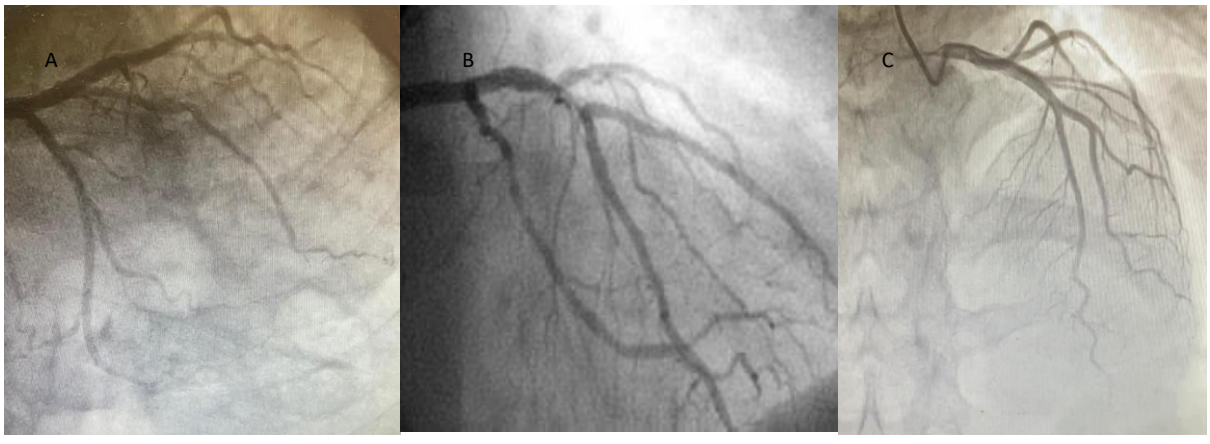


FIGURE 1(A)-CASE 1
FIGURE 1(B)-CASE 2
FIGURE 1[C]-CASE 3
HRCT SHOWING GROUND GLASS OPACITY

Figure 1 Showing HRCT of all three Cases



CORONARY ANGIOGRAPHY OF CASE 1(2A),CASE 2(2B) AND CASE 3 (2C) SHOWING NORMAL CORONARY ANGIOHRAPHY

Figure 2 showing normal coronary angiography in all three cases

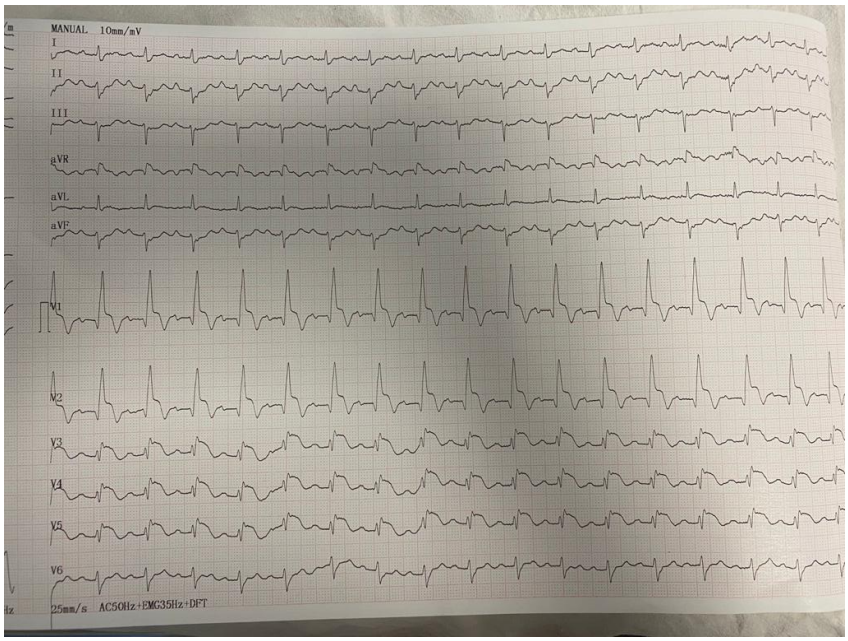


Figure 3 showing ECG of case 1

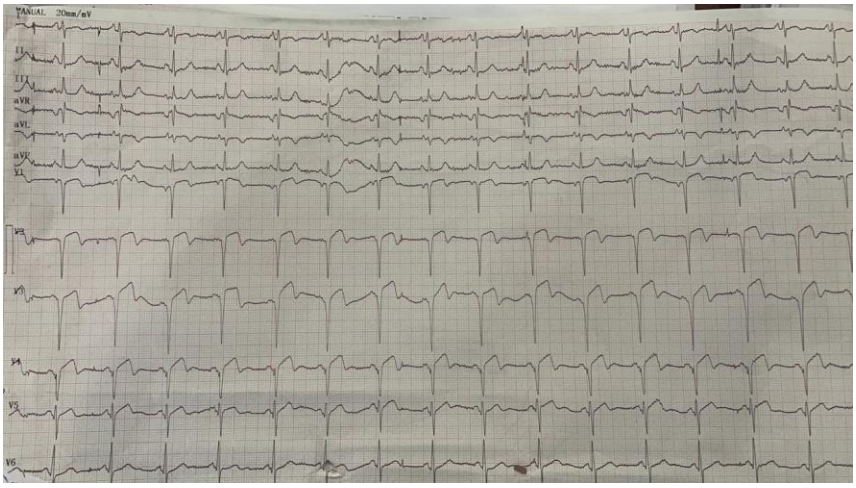


Figure 4 showing ECG of case 2

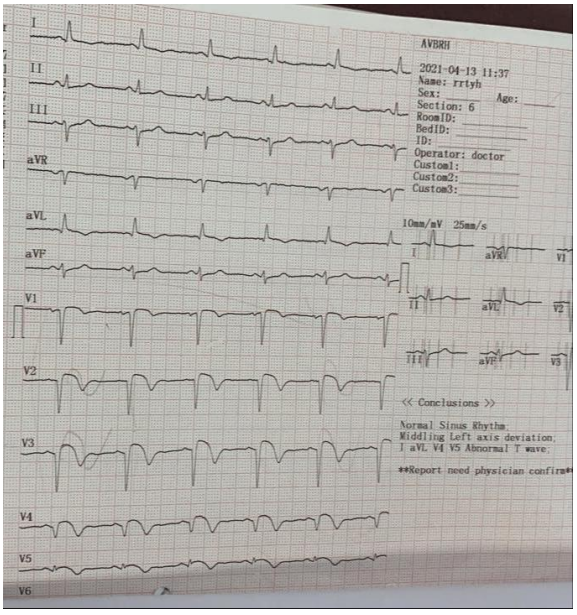


Figure 5 showing ECG of case 3

Table 1 Showing Lab investigations of all three cases

Lab Paramteter	Case 1 AGE-50Years SEX-MALE	Case 2 AGE-73 Years SEX-MALE	Case 3 AGE-39 Years SEX-MALE
CBC	Hb-10.3gm/dl MCV-80fl Platelet count-99000/dl WBC Count-14800/dl	Hb 10.6gm/dl, MCV:84fl, Platelet count 1.03/dl, WBC Count 15360/dl	Hb-11.6gm/dl, MCV:88fl, Platelet count 960000/dl, WBC Count 16400/dl
LFT	Total Protein-5.4gm/dl, Albumin3.0gm/dl, Globulin2.4gm/dl, aspartate aminotransferase 23 units/l , alanine aminotransferase 27 units/l, AlkanlinePhophatase 132 IU/l, Total Bilirubin :1.4mg/	Total Protein-6.0gm/dl, Albumin2.6gm/dl, Globulin3.4gm/dl, aspartate aminotransferase 34 units/l , alanine aminotransferase 33 units/l ,AlkanlinePhophatase121 IU/l, Total Bilirubin :1.9mg	Total Protein-5.1gm/dl, Albumin-1.9gm/dl, Globulin-3.2gm/dl, aspartate aminotransferase 33 units/l , alanine aminotransferase 28 units/l, AlkanlinePhophatase 110 IU/l, Total Bilirubin :0.9mg/dl,
KFT	Creatinine:0.7mg/dl, Urea44mg/dl, Sodium139mmol/l, Potassium -3.6mmol/l	Creatinine:0.9mg/dl, Urea22mg/dl, Sodium131mmol/l, Potassium – 3.9 mmol/l	Creatinine:1.2mg/dl, Urea43mg/dl, Sodium-133mmol/l, Potassium – 4.63mmol/l
CRP	92.0mg/dl	81.7mg/dl	71.9mg/dl
D-Dimer	0.49	0.48	0.44
Serum Ferritin	660ng/ml	750ng/ml	790ng/ml
HRCT Score CORAD	22/25 5	20/25 6	21/25 6
CKMB TROP I	33 POSITIVE	45 POSITIVE	65 POSITIVE

3. DISCUSSION

A Type 2 Myocardial Infarction is defined as Myocardial Infarction which is not a result of Coronary Artery Disease and is due to imbalance between oxygen supply and demand. In our case series we have reported 3 cases of patients who developed severe hypoxemia which resulted in increased oxygen demand of myocardial cells causing necrosis thereby leading to type 2 myocardial infarction (Hessel et al., 2008). It is essential to note that the coronary angiography was normal in all three patients who belonged to different age spectrum and were treated like type 2 myocardial infarction. While case 1 and 3 responded to oxygen therapy and improved and were discharged, case 2 had severe hypoxia which worsened further and ultimately did not survive. Also, there is a background of Sepsis in all three cases with ground glass opacity on HRCT. Ground Glass Opacity in bilateral lower lung field is a important characteristic finding of COVID19 pneumonia (Jain et al., 2021).

Due to sepsis there is cardiomyocyte death which can be resulted by complements, toxins and other myocardial depressants. Also, there is aggravation of adrenergic response at the level of cardiomyocyte due to beta adrenergic receptor down regulation. These Changes are modulated by Nitric Oxide and Cytokines released during inflammatory storm in COVID19 (Talwar et al., 2021). The Cytokine Storm witnessed in later stage of COVID19 might be a bug contributory factor is this pathophysiology of type 2 myocardial infarction as it has shown huge role in pathophysiology of stroke and other complications of COVID19 (Wanjari et al., 2020). Thus, through the medium of this case series we try to report world's first report on Type 2 myocardial infarction seen in COVID19 which remains to be a under reported entity.

4. CONCLUSION

The heterogenic nature of Type 2 Myocardial Infarction is a big challenge emerging during the challenging times of COVID19. Though type 1 myocardial infarction remains to be common complication during and after COVID19, Type 2 myocardial infarction has not been given much importance. We highlight the need of vigilance and prompt diagnosis of type 2 myocardial infarction by the physicians treating COVID19 to reduce the ongoing mortality of this deadly pandemic.

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We thank all the participants who have contributed in this Study.

Conflict of interest

The Authors have no conflicts of interest that are directly relevant to the content of this clinic-pathological case

Financial Resources

There are no financial resources to fund this study

Informed Consent

Informed Consent was obtained from the patient.

Author's contribution

All the authors contributed equally to the case report.

Data and materials availability

All data associated with this study are present in the paper.

REFERENCES AND NOTES

1. Alpert JS, Thygesen KA, White HD. Diagnostic and therapeutic implications of type 2 myocardial infarction: Review and commentary. Am J Med 2014; 127:105–8.
2. Hessel MHM, Atsma DE, van der Valk EJM. Release of cardiac troponin I from viable cardiomyocyte is mediated by integrin stimulation. Pflugers Arch 2008; 455:979–86.
3. Jain A, Talwar D, Kumar S. Spectrum of Respiratory Involvement in COVID 19 Era; An Overview. Indian J of Forensic Med & Toxicol 2020; 14(4):6593-9.
4. Pierpont GL, McFall EO. Interpreting troponin elevations: do we need multiple diagnoses? Eur Heart J 2009; 30:135-138.
5. Talwar D, Kumar S, Madaan S, Khanna S, Annadatha A. Intractable Singultus: Atypical presentation of COVID 19. Med Sci 2021; 25(111), 1183-1187
6. Thygesen JS, Alpert AS, Jaffe ML, Simoons BR, Chaitman HD. White, The Writing Group on behalf of the Joint ESC/ACCF/AHA/WHF Task Force for the Universal Definition of Myocardial Infarction Third universal definition of myocardial infarction Eur Heart J 2012; 33:2551-2567.
7. Wanjari AK, Ayush Dubey, Sourav Chaturvedi, Sunil Kumar. Young COVID 19 presenting as fatal subarachnoid hemorrhage: Association or chance? Med Sci 2020; 24(104), 2712-2715