Failure of Therapeutic Management in RBBB Myocardial Infarction Patients with Covid-19 Pneumonitis Admitted at Emergency Department of Tertiary Care Teaching Hospital a Case Series

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Abstract: Background: SARS-Cov-2(covid-19) infection is associated with Systemic Hyercoagulability and Thrombo-Embolic Complications. Anticoagulants play a key role in their management. Despite anticoagulants we have observed five case of RBBB MI in our ICU which co-relates with the thrombotic complication. And hence, Efficacy of the anticoagulants is debatable and requires meticulous APTT monitoring. Efficacy of thrombolysis is also not clear. A current guideline gives more emphasis on PCI over thrombolysis in RBBB MI patients. BUT unfortunate turn of events into catastrophe happened in these cases due to non-feasibility of cathlab interventions in covid-19 pandemic scenario. Material and Methods: This is A case series of five patients that were COVID-19 positive admitted to the ICU and Developed RBBB MI during their ICU stay despite on anticoagulant therapy. Data was collected retrospectively from hospital indoor records. Positive case of COVID-19 was confirmed by RT-PCR assay of nasopharyngeal or oropharyngeal swab specimen. Conclusion: Development of RBBB MI in covid-19 patients on anticoagulants co-relates with the thrombotic complication of SARS-CoV-2 infection and failure of the therapeutic management. PCI can be considered as a gold standard intervention compared to thrombolysis. BUT unfortunate turn of events into catastrophe happened in these cases due to non-feasibility of cathlab interventions in covid-19 pandemic scenario. [Mankodia D Natl J Integr Res Med, 2021; 12(6): 98-103]

Key Words: Failure of Therapeutic Management, RBBB MI, Covid-19 Pneumonitis

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Introduction: Severe acute respiratory distress caused by SARS-CoV-2 was declared as pandemic in March 2020 by World Health Organisation[1,2,3]. The infection caused by corona virus (covid-19) ranges from an asymptomatic carrier state to MODS / sepsis / ARDS resulting into high mortality within short period of hospitalization[4].

Moreover, the Covid-19 Associated Hypercoagulability leading to thrombo-embolic events is also seen in the patients but the exact mechanism is not very clear[5]. Sever Inflammation, Activation of coagulation cascade by the host defence system or cytokine storm may lead to formation of micro-thrombi into circulation. Moreover, Formation of the micro-thrombi into pulmonary circulation can further worsen the hypoxemia. Post-mortem examination of the lungs showed micro-circulatory clots in the patients who died of SARS-Cov-2[6,7]. Hence, to prevent thrombo-embolic events in covid-19 patient's anti-thrombotic agents play a key role in the management. LMWH has shown to reduce the formation of micro-thrombi and its prophylactic dosage was associated with lower 28 day's mortality and reduced hospital stay with better hospital outcome[1,2,3]. Dosing and efficacy of UFH v/s LMWH and newer anticoagulants is debatable. Hence, ideal regime for thrombo-prohylaxis is not yet Established[3].

RBBB Myocardial Infarction: RBBB is commonly seen in large antero-septal wall myocardial infarction where proximal LAD is occluded[11]. ECG suggests prolongation of QRS duration ≥ 120 ms, with rsr', rsR' or rSR' pattern in V1 or V2 chest leads[8]. New onset RBBB itself is an important independent risk factor for the adverse outcome amongst the in- hospital patients AND RBBB itself is associated with underlying large myocardial infection until proved otherwise.

Acute Myocardial infarction with RBBB itself is having poor prognosis and is treated mainly by primary PCI[8]. Previous studies have shown Benefits of thrombolysis in terms of reduction in size of clot, protection of LV function and decrease in mortality rate[9]. But current guidelines of European Society Of Cardiology

emphasis on primary percutaneous coronary intervention over thrombolysis[10]. The use of anticoagulants and DAPT (dual anti-platelets) is indicated in RBBB myocardial infarction patients unless contraindicated[11].

Case Series: **Case 1:** A 77-year-old male patient with COVID-19 positive RT-PCR report was admitted to a tertiary care teaching hospital with chief complaints of gabhramman, perspiration and chest pain since 3 days. Patient was known case of type 2 diabetes mellitus since 10 years on treatment.

He had positive risk factor of sedentary lifestyle. On admission his temperature was normal, heart rate was 90/min, blood pressure of 118/76 mmhg and respiratory rate of 22/min, spo2 of 93% on room air. On respiratory system bilateral lower zone crepitations were heard. No other abnormalities were seen in systemic examination. In view of tachypnoea and low oxygen saturation nasal prong 3l/min support was given.

On admission investigations- Hemoglobin: 12.1 gm/dl, Total counts: 16,000/cumm, platelet count was 4, 50,000/cumm. Renal function test and Liver function test was normal. Coagulation profile was within normal limit with D-Dimer >20 (<0.5: reference range). LDH: 660 U/L, Ferritin: 884 ng/dl, CRP: 210 mg/dl, trop I: 0.0001 (<0.1: reference range) and NT-proBNP: 46 pg/ml. 2D Echo was done bed side suggestive of LVEF: 55%.

Chest X-ray PA view suggestive of bilateral lower zone consolidation. HRCT chest: 14/25 score. ECG: sinus tachycardia with no ST-T changes.

Patient was treated with higher antibiotics, Inj. Remdesevir, steroids, antiplatelet, statins and anticoagulant in form of conventional heparin as infusion (in view of higher D-Dimer value). On the 5th day of hospital stay patient suddenly became tachypneic with respirator rate of 32/min along with perspiration and chest pain, so was put on bipap support. 12 lead ECG was taken suggestive of RBBB MI.

Urgently cardiology opinion was taken, bed side 2D Echo was done suggestive of LVEF-10-15% with global LV hypokinesia and thrombolysis was initiated with streptokinase. After 30 minutes ofstreptokinase infusion patient collapsed, was urgently intubated and CPR was started, but couldn't be revived after all resuscitative measures.

Case 2: A 57-year-old male patient with COVID-19 positive RT-PCR report was admitted to a tertiary care teaching hospital with chief complaints of cough with expectoration, fever and gabhramman since 5 days. Patient was known case of Type 2 diabetes mellitus since 15 years and Hypertension since 10 years on treatment.

He had sedentary lifestyle and was a chronic smoker. On admission his temperature was normal, heart rate was 118/min, blood pressure of 148/96 mmhg and respiratory rate of 32/min, spo2 of 73% on room air. On respiratory system bilateral air entry was equal with bilateral lower and mid zone crepitations were heard. No other abnormalities were seen in systemic examination. In view of tachypnoea and low oxygen saturation patient was urgently put on bipapsupport.

On admission investigations- Hemoglobin: 12.1 gm/dl, Total counts: 19,780/cumm, platelet count was 3,70,000/cumm. Renal function test and Liver function test was normal. Coagulation profile was within normal limit, with D-Dimer 9.8 (<0.5: reference range). LDH: 760 U/L, Ferritin: 704 ng/dl, CRP: 110 mg/dl, trop I: 0.0001 (<0.1: reference range) and NT-proBNP: 112pg/ml.

2D Echo was done bed side suggestive of LVEF: 50%. Chest X-ray PA view suggestive of bilateral lower zone haziness. HRCT chest: 22/25 score. ECG: AF with Fast Ventricular rate. Patient was treated with higher antibiotics, Inj. Remdesevir, steroids, antiplatelet, statins, anticoagulant in form of conventional heparin as infusion, anti-arrythmic drugs and Diuretics.

On the 2th day of hospitalisation patient was intubated and was put on mechanical ventilator.

On the 7th day of hospitalisation patient developed ventricular tachycardia with pulse.

Urgently synchronised cardiovesion was done and 12 lead ECG was taken which was suggestive of RBBB MI. Urgently cardiology opinion was taken, bed side 2D Echo was done suggestive of LVEF-10% with global LV hypokinesia and thrombolysis was initiated with streptoklinase. After 5 hours of streptokinase completion patient collapsed with PEA on the monitor. Urgently CPR

was started, but couldn't be revived after all resuscitative measures.

Case 3: A 67-year-old femle patient with COVID-19 positive RT-PCR report was admitted to a tertiary care teaching hospital with chief complaints of fever, cough without expectoration and breathlessness since 2 days. Patient was known case of type 2 diabetes mellitus since 5 years, hypertension since 10 years and Asthma since 5 years on treatment.

On admission his temperature was normal, heart rate was 80/min, blood pressure of 128/86 mmhg and respiratory rate of 18/min, spo2 of 95% on room air. On respiratory system expiratory wheeze was heard.

Urgently patient was nebulised in view of expiratory wheeze.No other abnormalities were seen in systemic examination. On admission investigations- Hemoglobin: 10.1 gm/dl, Total counts: 11,000/cumm, platelet count was 2, 80,000/cumm. Renal function test and Liver function test was normal. Coagulation profile was within normal limit, with D-Dimer 5.92 (<0.5: reference range). LDH: 260 U/L, Ferritin: 584 ng/dl, CRP: 58 mg/dl, trop I: 0.001 (<0.1: reference range) and NT-proBNP: 66 pg/ml. 2D Echo was done bed side suggestive of LVEF: 50%.

Chest X-ray PA view suggestive of prominence of broncho-vascular markings. HRCT chest: 9/25 score. ECG: normal. Patient was treated with steroids, antiplatelet, statins, anticoagulant in form of conventional heparin as infusion and symptomatic medications were given. On the 3th day of hospital stay patient suddenly became tachypneic with respirator rate of 22/min. So was put on 10l/min oxygen mask support.

Repeat investigations were sent which were as follows: Hemoglobin: 9.8 gm/dl, Total counts: 15,000/cumm, platelet count was 1, 89,000/cumm. Renal function test and Liver function test was normal. Coagulation profile was within normal limit, with D-Dimer 10.92 (<0.5: reference range). LDH :860 U/L, ferritin :984 ng/dl, CRP :258 mg/dl, trop I: 0.158 (<0.1: reference range) and NT-proBNP: 1680 pg/ml. Chest X-ray PA view suggestive of bilateral diffuse opacities. Higher antibiotics and Inj. Remdesevir was added. On the 5th day the condition of the patient deteriorated and was put on bipapsupport. On the 7th day patient was intubated in view of tachypnea and non-maintenance of saturation on bipap support.

On 7th day D-Dimer came out to be >20, anticoagulants were continued and APTT was maintained 2 times the upper normal limit. On 11th day patient suddenly developed pulseless ventricular tachycardia. Urgently patient was Given DC shock followed by CPR. Patient revived after 3 Cycles of CPR.

Following that 12 lead ECG was taken suggestive of RBBB MI. Urgently cardiology opinion was taken, bed side 2D Echo was done suggestive of LVEF-10% with global LV hypokinesia and thrombolysis was initiated with streptoklinase.

After 15 minutes of thrombolysis initiation patient again collapsed with PEA on the monitor. Urgently CPR was started, but couldn't be revived after all resuscitative measures.

Case 4: A 37-year-old male patient with COVID-19 positive RT-PCR report was admitted to a tertiary care teaching hospital with chief complaints of high grade fever since 7 days and breathlessness since 2 days, Chronic Alcoholic. Patient had taken treatment from some local hospital for 5 days (No documents available). On admission his temperature was 101 F, heart rate was 129/min, blood pressure of 118/72mmhg and respiratory rate of 32/min, spo2 of 60% on room air. On respiratory system right sided air entry was decreased with left side basal crepitations.

No other abnormalities were seen in systemic examination. In view of tachypnoea and increasing oxygen requirement patient was given HFNC support (60L/100%). On admission investigations- Hemoglobin: 13.1 gm/dl, Total counts: 19,000/cumm, platelet count was 3, 80,000/cumm. D-Dimer 18.92 (<0.5: reference range). LDH :760 U/L, ferritin :984 ng/dl, CRP: 568 mg/dl, trop I: 0.001 (<0.1: reference range) and NT-proBNP: 114pg/ml. 2D Echo was done bed side suggestive of LVEF: 50%.

Chest X-ray PA view suggestive of right sided mild pleural effusion and left lower lobe consolidation. HRCT chest: 23/25 score. ECG: sinus tachycardia with T wave inversion in lead V1 to V6. Patient was treated with antibiotics, inj. Remdesevir, steroids, antiplatelets, statins, anticoagulant in form of conventional heparin as infusion and symptomatic medications were given. On the 2th

day of hospital stay patient suddenly became tachypneic with respirator rate of 42/min. So was put on bipap support fio2 100%.

On the 3rd day of hospitalisation patient developed severe retrosternal chest pain, perspiration and hypotension. 12 lead ECG was taken suggestive of RBBB MI and Trop I: >50. Urgently central venous access was taken and inotropic support was started.
After 2 hour's inotropic support has to be maximised due to refractory cardiogenic shock.

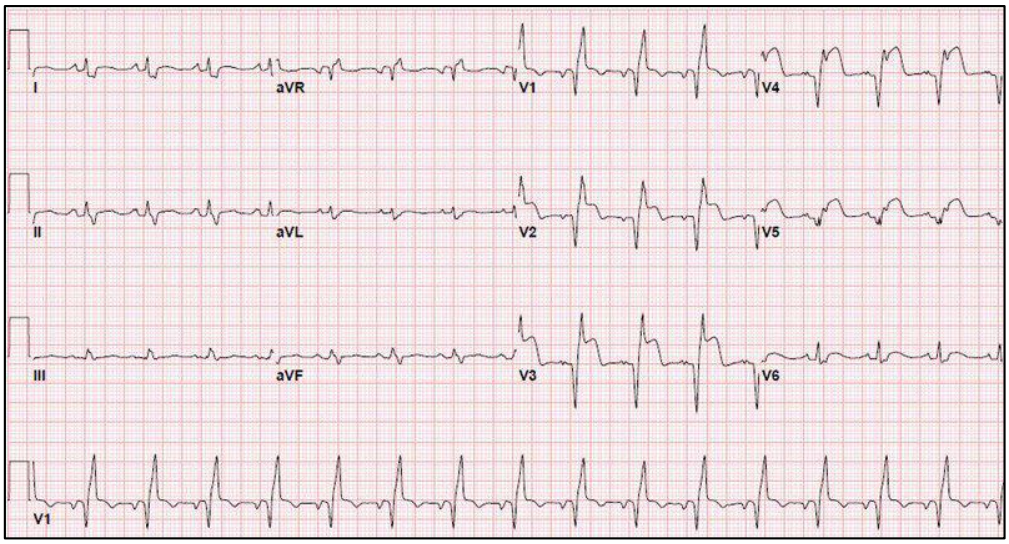
Case 5: An 86-year-old male patient with COVID-19 positive RT-PCR report was admitted to a tertiary care teaching hospital with chief complaints of low grade fever since 7 days followed by generalised weakness, cough with expectoration and breathlessness. Patient is known case of type 2 diabetes mellitus, hypertension since 30 years on treatment and ischemic heart disease since 10 years on treatment. Chronic Alcoholic, chronic smoker and chronic tobacco chewer. On admission his temperature was normal, heart rate was 108/min, blood pressure of 90/52 mmhg and respiratory rate of 32/min, spo2 of 60% on room air. On respiratory system bilateral coarse crepitation was heard upto the midzone.

No other abnormalities were seen in systemic examination. Patient's GCS was <7 and was urgently intubated and kept on mechanical ventilator. Central line was inserted and inotropic support was started. On admission investigations- Hemoglobin: 9.1 gm/dl, Total counts: 33,000/cumm, platelet count was 1, 80,000/cumm. Renal function test and Liver function test was altered. Coagulation profile was also altered, with D-Dimer >20 (<0.5: reference range). LDH: 1150 U/L, ferritin: 988 ng/dl, CRP: 768 mg/dl, trop I: >50 (<0.1: reference range) and NT-proBNP: >5000pg/ml. 2D Echo was done bed side suggestive of LVEF: 20%, global LV hypokinesia with minimal pericardial effusion.

HRCT chest: 20/25 score with cardiomegaly and congestive changes. ECG: RBBB MI. Urgently cardiology opinion was taken and patient was treated with higher antibiotics, inj. Remdesevir, dual antiplatelets, statins and anticoagulant in form of conventional heparin as infusion. In view of persistent hypotension despite maximum inotropic support thrombolysis was not done. On the 2th day of hospital stay patient suddenly collapsed with PEA on the monitor. Urgently CPR was stated, but couldn't be revived after all resuscitative measures.

All the five patients had received the anticoagulant in the form of conventional heparin as an infusion 1,000 IU 1 hourly before the development of RBBB MI. Cardiology opinion was taken; bed side 2D Echo was done suggestive of LVEF-18% with global LV hypokinesia and due to hypotension thrombolysis could not be done. Inj. Heparin was continued. On the 4th day elective intubation was performed and patient was put on mechanical ventilator. Few hours later patient collapsed with PEA on the monitor. Urgently CPR was started, but couldn't be revived after all resuscitative measures.

Image 1: ECG Showing RBBB Anterior Wall Myocardial Infarction.



Discussion: Hospitalised covid-19 patients should always be considered as having high risk of thrombo-embolic events. Hence, thrombo-prophylxis should always be considered. Parentral anticoagulants i.e. UFH (unfractionated heparin) and LMWH (low molecular weight heparin) are the preferred ones. Ideal dosing regime is not established yet[3].

The efficacy of the anticoagulants is monitored by the serial APTT levels monitoring. Sometimes, it may lead to bleeding complications also. The prophylactic-dose anticoagulation, mainly LMWH, was associated with lower 28-day mortality in elevated D-dimer compared to nonusers[3].

When anticoagulated COVID-19 patients are admitted to hospital, especially to an ICU achieving UFH therapeutic a PTT targets is another issue[3].Non achievement of the APTT levels at the therapeutic dose can doubt the efficacy and failure of anticoagulants. Higher dose can be considered to achieve the levels but the risk of bleeding also increases. Development of RBBB MI itself is having the poor outcome.

But when it is developed in COVID-19 patients on anticoagulants is due to the lower efficacy of anticoagulants. Efficacy of Thrombolysis in RBBB MI is also debatable. Studies have shown that when the bundle branch block is relieved on thrombolysis the prognosis is favorable. But, still the role of thrombolysis is not clear.

Thrombolysis with streptokinase was done in three patients while in the remaining due to cardiogenic shock it was not given. Streptokinase can itself cause hypotension hence its role is also not clear. In our ICU Setup streptokinase is easily available but efficacy is doubtful in RBBB MI.

According to the ESC (European society of cardiology) primary PCI is considered as gold standard as compared to thrombolysis. In-hospital mortality of patients with AMI and RBBB is highest from all ECG presentations of AMI.

Restoration of coronary flow by primary PCI may lead to resolution of the conduction delay on the discharge ECG[11]. Due to non-feasibility of cathlabs in covid-19 pandemic scenario and failure of therapeutic management with anticoagulants and thrombolysis the catastrophic event i.e. RBBB MI can occur resulting into higher mortality. In future IABP (intra-aortic balloon pump) can be considered in the patients if cardiogenic shock is present. IABP and PCI are the emergency procedures in RBBB MI that can cause a favourable outcome in future.

Hence, the overall prevalence of the ACS in covid-19 pandemic is underestimated because of non-feasibility of cathlabs and respiratory symptoms are cardinal which sometimes overlaps with the symptoms of ACS[12,13,14].

Conclusion: Development of RBBB MI in covid-19 patients on anticoagulants co-relates with the thrombotic complication of SARS-CoV-2 infection and failure of the therapeutic management. Efficacy of thrombolysis is also not very clear.

PCI can be considered as a gold standard intervention compared to thrombolysis. BUT unfortunate turn of events into catastrophe happened in these cases due to non-feasibility of cathlab interventions in covid-19 pandemic scenario.

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