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Original Article

Management of Acute Coronary Syndrome in COVID-19 Patients – A Single-Center Study

G Selvarani[1], T R Hemanath[2], S Sathish Kumar[2], S R Veeramani[3], S Balasubramanian[4], M Natarajan[5], R Prabhakaran[6]

[1]Associate Professor, Department of Cardiology, Madurai Medical College, Madurai, Tamil Nadu, India, [2]Assistant Professor, Department of Cardiology, Madurai Medical College, Madurai, Tamil Nadu, India, [3]Professor and Former Head, Department of Cardiology, Madurai Medical College, Madurai, [4]Professor and Head, Department of Cardiology, Madurai Medical College, Madura, [5]Professor and Head, Department of Medicine, Madurai Medical College, Madurai, [6]Professor and Head, Department of Thoracic Medicine, Madurai Medical College, Madurai, Tamil Nadu, India

Abstract

Introduction: In coronavirus disease 2019 (COVID-19) pandemic, many patients suffered acute coronary syndrome (ACS) which includes ST-segment elevation myocardial infarction (STEMI), non-STEMI (NSTEMI), unstable angina, and sudden cardiac death. There is increased risk of acute myocardial infarction with newly diagnosed COVID-19 compared to non-infective controls.

Purpose: The purpose of the study was to study the incidence of ACS in COVID-19 patients admitted from April 2021 to March 2022 to compare outcome with ACS cases in non-COVID intensive care unit (ICU) of same center and to come out with guidelines for the management of ACS with COVID-19 to reduce mortality in centers without dedicated COVID Cath Labs.

Materials and Methods: Population of the study comprises adult patients presenting to COVID wards with COVID 19 and ACS and patients with ACS without COVID of the same hospital for the period of April 2021–March 2022.

Results: We had 62 ACS cases, who were COVID-19 positive. Among them, 37 cases were STEMI, 20 cases of Unstable Angina and 5 cases of NSTEMI. 33 patients in COVID wards were lysed, no STEMI patients underwent primary PCI and PIT. In the same period, we had 1541 ACS patients admitted in Coronary Care Unit (Non-COVID wards). Among them, 1496 cases were STEMI, 24 cases had Unstable Angina and 21 cases had NSTEMI. Among those 1541 ACS cases, 1125 patients underwent thrombolysis, 86 patients underwent primary PCI and 112 patients underwent PIT. Among 62 ACS cases in COVID wards, there were 10 STEMI deaths, 3 Unstable Angina deaths and 1 NSTEMI death. Among 1541 ACS cases in Non-COVID wards, 231 STEMI deaths and no death in Unstable Angina and NSTEMI.

Conclusion: Role of thrombolytic therapy alone in COVID STEMI in reducing mortality is non-inferior to PPCI and pharmacoinvasive PCI in non-COVID STEMI. Mortality in COVID STEMI is also because of severe COVID infection. Incidence of unstable angina is higher in COVID patients.

Key words: Acute coronary syndrome, Coronavirus disease 2019, Non-COVID, Thrombolysis

INTRODUCTION

In the coronavirus disease 2019 (COVID-19) pandemic,[1] patients suffered from acute coronary syndrome (ACS) which includes ST-segment elevation myocardial infarction (STEMI), non-STEMI (NSTEMI), unstable angina, and sudden cardiac death.

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Acute myocardial injury in COVID-19 patients[2] has multiple mechanisms, myocardial ischemia caused by systemic hypoxia, in setting of severe acute respiratory distress syndrome, multiple thrombosis, coronary spasm, systemic inflammatory response due to cytokine storm, and vasculitis like vessel damage is likely triggers that lead to rupture of atherosclerotic plaque. Systemic Inflammation leads to activation of cytokines initiating rupture of pre-existing atherosclerotic plaque. There is imbalance between myocardial oxygen demand and supply due to respiratory failure, tachyarrhythmia and sepsis. COVID-19 patients have increased thrombotic tendency due to endothelial damage, dehydration and increased cytokines. All these contribute to the increased risk for Acute Coronary Syndrome.

Corresponding Author: Dr. G Selvarani, Silovam Hospital, 3/268, New Natham Road, Oomachikulam, Thirumalpuram PO, Madurai, Tamil Nadu, India.

The clinical peculiarities of ACS in COVID-19 patients[3] are that the clinical picture of ACS is masked by course of the infectious disease itself.[4] The diagnosis of ACS may be delayed and medical personnel should focus on entire complex of clinical manifestations and examination data such as electrocardiograph (ECG), echocardiography, and enzyme levels and other imaging modalities.

In COVID wards, health-care providers were doing duties in shifts for 6 h with strict PPEs. All health-care providers were instructed to treat ACS cases using telemedicine[5] in most of the centers. There was confusion in diagnosing ACS cases due to ST, T changes associated with COVID which affected multiple systems. Even after diagnosis, the management options are limited due to non-availability of dedicated Cath Labs. Thrombolytic therapy with streptokinase is preferred modality in such circumstances. The survival rate following thrombolytic therapy is comparable with invasive strategy. In many cases, Covid dominates ACS patient has to fight with two major killer diseases.[6] The survival depends on the multiple factors such as immune status, comorbid conditions, and availability of management strategies. Individualized, COVID cardiac team approach is needed for every patient.

MATERIALS AND METHODS

Study Population

It comprises adult patients presenting to COVID wards with ACS. Patients with COVID having chest pain, dyspnea, palpitation confirmed myocardial infarction (MI) by universal definition of MI through enzymes, ECG, and echo were included in the study. The demographic profile ACS patients admitted in COVID wards and their management and in-hospital mortality were collected. Non-COVID ACS data were collected for same period, and compared the in-hospital mortality.

Study Period

During COVID-19 pandemic, between April 2021 and March 2022 (second wave).

Statistical Analyses of the Data

Statistical analyses of the data are conducted using the software IBM SPSS 20.0 version. Statistical inference was derived using Chi-square test.

Inclusion Criteria

- Patients present with ACS – STEMI, NSTEMI, and unstable angina
- Patients presenting with acute MI as diagnosed by electrocardiogram defined as new ST elevation at the J point in at least 2 contiguous leads ≥2 mm in men or ≥1.5 mm in women in leads V2–V3 and/or of ≥1 mm in other contiguous chest or limb leads admitted in the COVID wards
  - With real-time reverse transcription-polymerase chain reaction (RT-PCR) test positivity
  - With or without pneumonia ground-glass opacity (GGO) by computed tomography (CT) chest
  - COVID pneumonia by CT chest GGO with or without RT-PCR positivity
- Patients of age equal to or >18 years of both sexes.

Exclusion Criteria

- All non-COVID patients with ACS
- Patients with pseudo-infarctions (*).

(*)There have been reports of myocardial injury occurring in patients who have tested positive for COVID-19 with evidence of troponin leak and elevation, and these patients may initially present with a pseudo-infarct pattern in ECG. Other conditions for pseudo-infarctions are given below:

(1) Acute pericarditis, (2) myocarditis, (3) early repolarization pattern, (4) hyperkalemia, (5) Brugada pattern, (6) Ebstein anomaly, (7) epsilon wave in arrhythmogenic right ventricular dysplasia, (8) Osborn wave of hyperthermia, (9) in the left ventricular hypertrophy, in hypertrophic cardiomyopathy (HCM), (10) acute pancreatitis, (11) intracranial hemorrhage, (12) subdural hematoma, (13) in pulmonary emphysema, patients with pneumothorax and pulmonary embolism, (14) myocardial fibrosis in patients with dilated cardiomyopathy, progressive muscular dystrophy, Friedreich's ataxia, scleroderma, amyloidosis, and primary and metastatic tumors of the heart, (15) QS deflections are often seen in the right precordial leads in patients with complete left bundle branch block in the absence of myocardial infarction, and (16) the delta waves in Wolff-Parkinson-White syndrome.

RESULTS AND ANALYSIS

COVID ACS

During COVID pandemic 2nd wave, between April 2021 and March 2022,[7] there were 18,104 admissions in COVID ward. Out of them, 62 cases were admitted with ACS (0.34%). Among the 18,104 cases [Table 1], there were 427 deaths (2.36%). Among 62 cases of COVID ACS, 37

Table 1: The no. of patients admitted and no. of mortality in COVID wards

COVID	No. of patients
IP	18,104
Death	427

COVID: Coronavirus disease

cases (60%) were STEMI, 20 cases were Unstable Angina (32%) and 5 cases were NSTEMI (8%) [Chart 1]. Out of 62 COVID ACS cases, 14 cases died (22.6%). In-hospital mortality of COVID ACS patients comes as 0.08%, that is, 8 patients per 10,000 cases died due to COVID ACS. In-hospital mortality of STEMI, NSTEMI, and unstable angina for the admitted (18,104) cases is 0.06%, 0.006%, and 0.02% [Table 2], respectively. In-hospital mortality of STEMI, NSTEMI, and unstable angina among 427 deaths was 2.3% [*n*-10], 0.2% [*n*-1], and 0.7% [*n*-3], respectively. COVID disease was either in the form of seropositivity or COVID pneumonia with or without seropositivity. Male: female ratio for COVID ACS was 0.73:0.27 (45 male + 17 female). There were no female cases with ACS below 50 years with COVID-19.[8] Incidence of ACS is highest among 60–69 years with COVID.

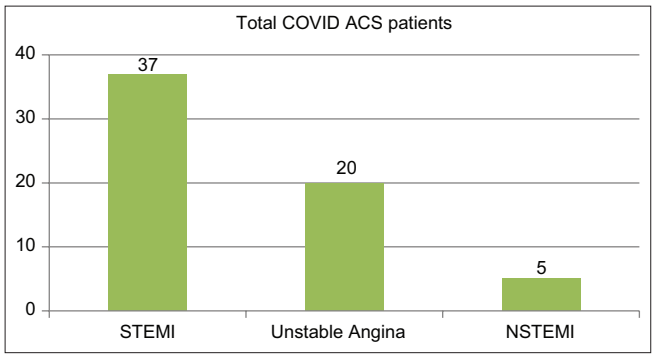


Chart 1: The total no. of COVID ACS patients which includes STEMI, unstable angina, and NSTEMI

Table 2: The no. of COVID ACS patients and their mortality among COVID In-patients (18,104 cases)

COVID ACS	ICU	In % (for 18,104 cases)	Death	In % (for 18,104 cases)
STEMI	37	0.2	10	0.06
NSTEMI	5	0.03	1	0.02
Unstable angina	20	0.1	3	0.006
Total	62	0.34	14	0.08

STEMI: ST-segment elevation myocardial infarction, NSTEMI: Non-STEMI, ICU: Intensive care unit, ACS: Acute coronary syndrome, COVID: Coronavirus disease

Table 3: The percentage of COVID ACS patients with comorbidities

Comorbidities	No. of patients (In %)
DM	35.5
HTN	27.4
CKD	1.6
Obesity	2.5
Dyslipidemia	5
Smoking	24.2
Alcoholism	17.8
Sedentary lifestyle	15
Anxiety and stress	5
Depression	1

ACS: Acute coronary syndrome, COVID: Coronavirus disease, DM: Diabetes mellitus, HTN: Hypertension, CKD: Chronic kidney disease

Comorbid conditions in ACS patients [Table 3] were diabetes mellitus (DM) – 35.5%, hypertension (HT) – 27.4%, and chronic kidney disease – 1.6%. About 2.5% were obese with body mass index ≥30%. Smoking was a risk factor in 24.2% of male cases. About 17.8% were alcoholics. Sedentary lifestyle was present in 15% of cases. Anxiety, depression, and stress were present in 5% of cases. Dyslipidemia was present in 5% of cases.

It is observed that in-hospital mortality of COVID ACS cases had increased with comorbid conditions [Table 4]. Half of them were diabetic. About 28.6% of them had HT and current smoking history. In-hospital stay of death cases was analyzed. Half of them (7 out of 14) died within 24 h. Mostly death occurred between 2 PM and 8 PM (50%) followed by 2 AM and 8 AM (42.9%). Overall death occurred in the age group between 60 and 69 years [Chart 2]. Mortality is high in males than females. COVID ACS in-hospital mortality is high (14 out of 62, i.e., 22.6%), when compared to non-COVID ACS in-hospital mortality in the same period of time. According to vaccination status among 62 COVID ACS patients, 9 had 1st dose, 21 had 2nd dose, and 32 were not vaccinated [Table 5].

Table 4: The co-morbidities and COVID-related conditions of COVID ACS patients who have died

Comorbidities and COVID-related conditions	Total (%)
Comorbidities	
DM	7 (50)
HTN	4 (28.6)
Smoking	4 (28.6)
CT – chest findings (in involvement)	
Mild	4 (28.6)
Moderate	4 (28.6)
Severe	6 (42.9)
In-hospital stay	
<12 h	3 (21.4)
12–24 h	1 (7.1)
24–48 h	2 (14.3)
Time of death	
8 am–2 pm	1 (7.1)
2 am–8 pm	7 (50)
8 am–2 am	0 (0)
2 am–8 am	6 (42.9)

CT: Computed tomography

Table 5: The vaccination status of COVID ACS patients

Vaccination	No. of patients
1st dose	9
2nd dose	21
Not vaccinated	32
Total	62

ACS: Acute coronary syndrome, COVID: Coronavirus disease

Non-COVID ACS

In coronary care unit, 1541 cases were admitted with ACS [Table 6]. STEMI, NSTEMI, and unstable angina were 1496 (97%), 21 (1.36%), and 24 (1.56%), respectively. Out of 1541 cases of ACS, 231 (15.4%) cases died due to MI complications. The in-hospital mortality of STEMI, NSTEMI, and unstable angina was 15.4% (231 cases), 0%, and 0%, respectively. Management in non-COVID ACS cases includes primary percutaneous coronary intervention (PPCI), thrombolysis, and pharmacoinvasive therapy (PIT). In spite of all three modalities of therapy, the in-hospital mortality is high due to STEMI (15.4%) alone.

COVID STEMI

STEMI[9] occurred between the age group of 50 and 59 years (9 males + 4 females) followed by 60–69 years (10 males + 2 females) [Chart 3]. Median delay for STEMI cases was between 6 h and 12 h in majority of cases (15 cases) [Chart 4]. Out of 37 STEMI cases, 28 were male and 9 were female with male: female ratio of 0.76:0.24. Among 37 STEMI cases, [Chart 5] 25 received thrombolytic therapy with streptokinase (SK), eight cases were referred after lysis for further management. Four cases were not lysed because of late arrival to hospital. Late arrival in the pandemic situation is multifunctional like fear of acquiring severe infection and silent myocardial infarction due to atypical symptoms such as shortness of breath and palpitation.

Two cases were received in cardiogenic shock. Among them, one had anterior wall myocardial infarction (AWMI) and another had inferior wall myocardial infarction (IWMI).

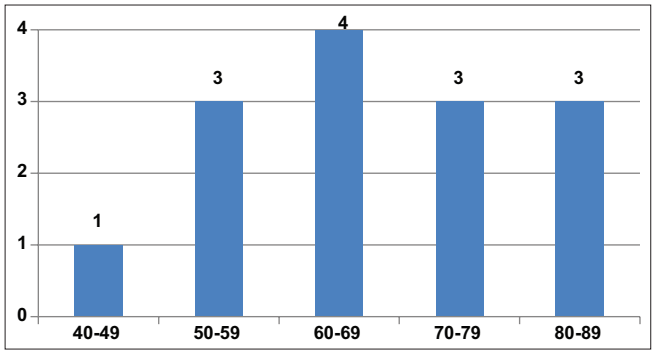


Chart 2: The mortality of COVID ACS patients according to their age group

AWMI case was lysed with SK and IWMI case was not lysed because of late arrival (>48 h). All STEMI cases were thrombolysed if they come in 12 h–24 h. Others were heparinized using low-molecular-weight heparin (LMWH) (enoxaparin 40 mg twice a day).

Since there is no dedicated COVID Cath Lab, we treated all STEMI cases with pharmacotherapy only. Only one patient underwent percutaneous transluminal coronary angioplasty (at private hospital) elsewhere following thrombolysis in our center (pharmacoinvasive PCI). Primary PCI could not be done as we mentioned already about lack of dedicated COVID Cath Lab [Table 7].

During lysis in COVID ICU, one patient developed ventricular tachycardia (VT) and another had ventricular fibrillation (VF). Both cases were direct current cardioverted. Among STEMI cases, nine patients had severe lung involvement with GGO of > 50% of lung fields with CORADS 5. Out of 37 STEMI, 10 cases died [Male-7 and Female-3]. The in-hospital mortality rate was higher with severe lung involvement. In-hospital mortality of COVID STEMI cases (27%) is high and overestimated, because six cases died of severe COVID pneumonia *per se*.

Non-COVID STEMI

Among 1496 non-COVID STEMI cases, [Table 7] thrombolysis was done to 1125 cases (75%), PPCI was done to 86 cases (5.7%), and PIT was offered to 112 cases (7.5%). Out of 1496 STEMI cases in non-COVID ICU, 231 deaths (in-hospital mortality – 15.4%). In spite of all three modalities of therapy, the in-hospital mortality was high (15.4%) in non-COVID STEMI when comparing with in-hospital mortality of COVID STEMI cases (27%).

COVID NSTEMI

Among 62 ACS cases, five cases – three males and two females had NSTEMI [Chart 6]. Four cases were between 50 and 60 years; one case was between 70 and 80 years. Diabetes and HT were the risk factors among NSTEMI cases. All NSTEMI were heparinized. One patient died due to severe COVID pneumonia. In-hospital mortality rate is high (one out of five) (i.e., 20%) in comparison with in-hospital mortality rate among NSTEMI cases admitted in non-COVID ICU (0 out of 25) (i.e., 0%) [Table 6].

Table 6: The total number of ACS patients in COVID and non-COVID ICUs and their death rate

Types of ACS	Total patients		Death		Death rate (in %)	
	COVID ICU	Non-COVID ICU	COVID ICU	Non-COVID ICU	COVID ICU	Non-COVID ICU
STEMI	37	1496	10	231	27	15.4
NSTEMI	5	21	1	0	20	0
Unstable angina	20	24	3	0	15	0
Total	62	1541	14	231	22.6	15

STEMI: ST-segment elevation myocardial infarction, NSTEMI: Non-STEMI, ICU: Intensive care unit, ACS: Acute coronary syndrome

Non-COVID NSTEMI

Non-COVID NSTEMI cases were 21 out of 1541 cases (1.4%). They were given heparin and guideline directed medical therapy followed by invasive therapy, that is, coronary artery angiography (CAG) and procedures. There were no deaths in non-COVID NSTEMI cases.

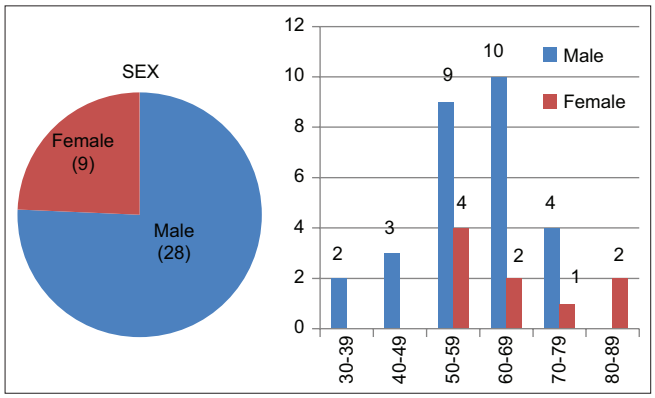


Chart 3: The number of STEMI patients in COVID wards according to sex and age criteria

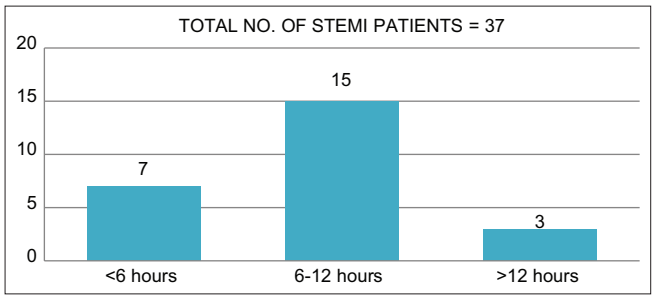


Chart 4: The duration of median delay for the COVID STEMI patients

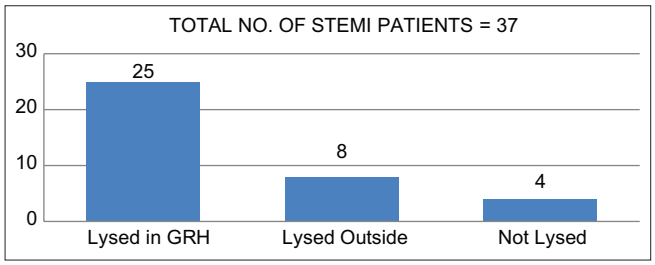


Chart 5: The thrombolysis status of STEMI patients in COVID

Table 7: The no. of STEMI patients undergone lysed, PPCI, and PIT in COVID and non-COVID wards

No. of STEMI	Management methods	No. of cases and (%)
COVID wards (37)	LYSED	37 (100)
	PPCI	0 (0)
	PIT	1 (2.7)*
Non-COVID wards (1496)	LYSED	1125 (75.2)
	PPCI	86 (5.75)
	PIT	112 (7.5)

(*) One patient underwent PIT privately, STEMI: ST-segment elevation myocardial infarction, PPCI: Primary percutaneous coronary intervention, PIT: Pharmacoinvasive therapy, STEMI: ST-segment elevation myocardial infarction

COVID Unstable Angina

There were 20 unstable angina cases, [Chart 7] 14 males and 6 females (male: female ratio – 7:3). Highest incidence occurred between 50 and 59 years of age. ST and T with changes were present in all cases and troponin levels were elevated in 50% of the cases. Typical angina with ST and T changes with elevated troponin levels was treated as unstable angina [Figure 1]. Those with diffuse ST and T changes [Figure 2] were categorized as myopericarditis, and they were followed up with echo, serial ECGs, and troponin. Cardiac magnetic resonance imaging would have thrown light to diagnose acute myocarditis and myopericarditis [Figure 3]. Eleven such cases were diagnosed in the same duration of our study.

All unstable angina cases were treated with LMWH (enoxaparin 40 mg IV twice a day). There were 3 deaths (15%) among unstable angina patients with COVID. This indicates that severe COVID contributed for increased in-hospital mortality in unstable angina.

Non-COVID Unstable Angina

Non-COVID unstable angina cases were 24 out of 1541 cases (1.6%) [Table 6]. They were managed with heparin-based therapy followed by invasive therapy, that is, CAG and procedures. There were no deaths in non-COVID unstable angina cases.

DISCUSSION

Since severe acute respiratory syndrome coronavirus 2 is a novel virus, there were no proper guidelines from randomized controlled clinical trials and studies to give[10] management protocols for ACS patients with COVID-19.[11] Many private hospitals were closed and there was shortage of workforce of doctors and staffs, since they were diverted to work in COVID wards in shift basis. Since there is no dedicated Cath Lab, primary PCI and pharmacoinvasive PCI could not be offered to COVID ACS patients. Many of them came late with evolved MI with poor left ventricle (LV) function. In these circumstances, we offered all STEMI cases (who were fit to receive thrombolytic therapy) thrombolysis with SK. All NSTEMI and unstable angina cases were treated with conservative medical therapy [Table 8]. LMWH was given intravenously 40 mg twice a day dose to have adequate anti coagulation without producing major bleeding. Aspirin (150 mg), Clopilet (75 mg), and Atorvastatin (80 mg) were given to all ACS cases unless contraindicated. Angiotensin-converting enzymes, angiotensin receptor blockers, and beta-blockers were avoided in some cases due to respiratory

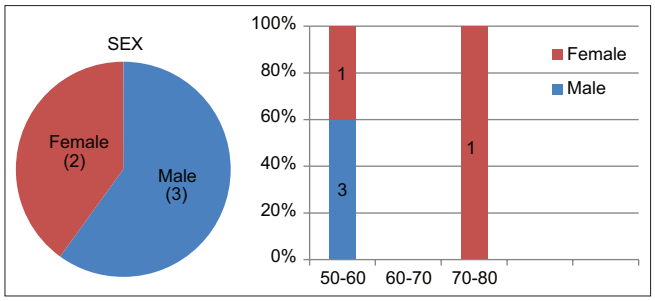


Chart 6: The number NSTEMI patients in COVID wards according to sex and age criteria

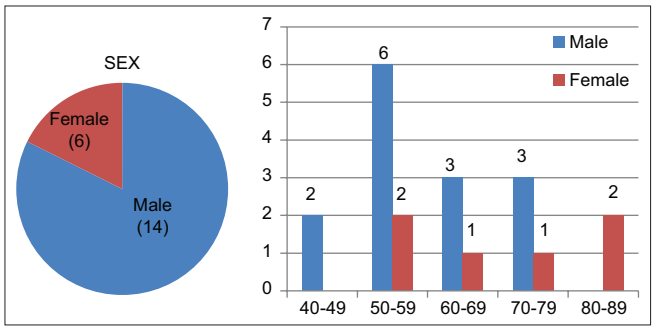


Chart 7: The number of unstable angina patients in COVID wards according to sex and age criteria

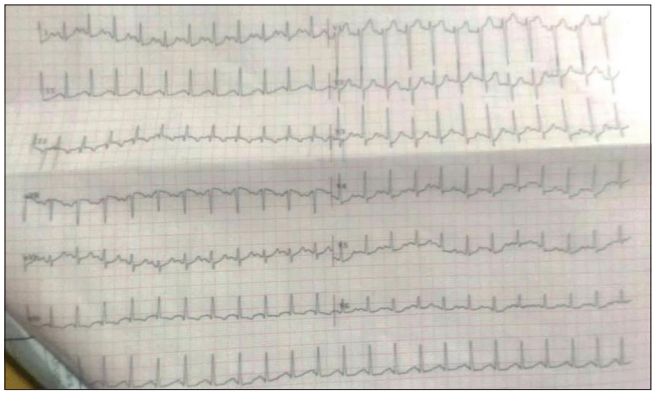


Figure 1: The ECG of COVID ACS patient with sinus tachycardia with ST depression

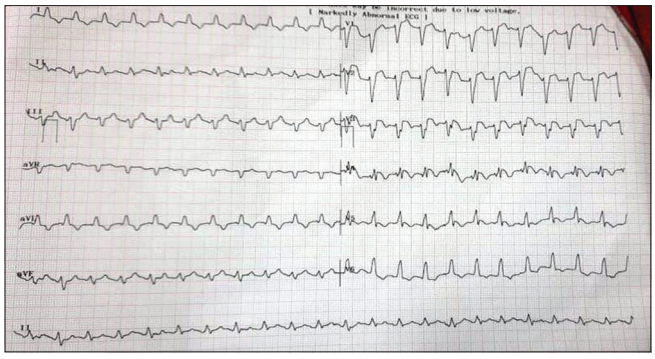


Figure 2: The ECG of COVID ACS patient with rheumatoid arthritis showing ST changes and 1st degree AV block

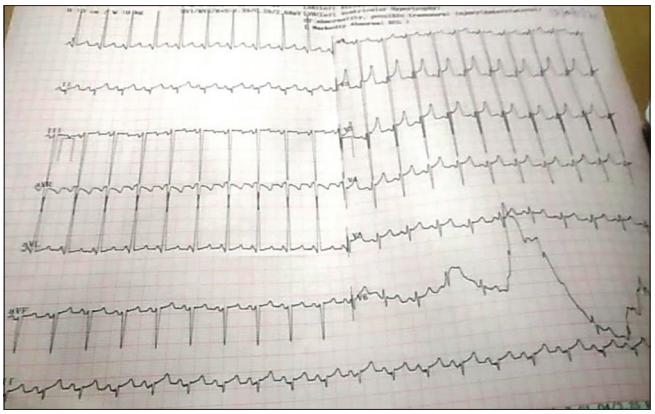


Figure 3: The ECG of a patient with myopericarditis

Table 8: The medical management protocol for COVID ACS patients

STEMI	NSTEMI	Unstable angina
Streptokinase	Heparin	Heparin
Heparin	Aspirin	Aspirin
Aspirin	Clopidogrel	Clopidogrel
Clopidogrel	Atorvastatin	Atorvastatin
Atorvastatin	Beta-blockers*	Beta-blockers*
ACE inhibitors*	ACE inhibitors*	ACE inhibitors*
Beta-blockers*		

(*) Used depending on the risk benefit ratio of the cases, STEMI: ST-segment elevation myocardial infarction, NSTEMI: Non-STEMI

distress due to severe lung involvement. Ivabradine used to reduce heart rate in sinus tachycardia [Figure 4] cases and also as a part of anti-failure management. Furosemide used with caution to avoid dehydration and electrolyte loss. Angiotensin receptor neprilysin inhibitor and sodium-glucose cotransporter-2 inhibitors were not used. Other management as per standard guidelines of ACS was followed. The outcome compared with non-COVID ACS cases who were admitted in the same period. In spite of interventional therapy, there is not much difference in the mortality rate comparing to thrombolytic therapy alone. Patients with MI and COVID died not only because of MI but also because of severe lung involvement.

During lysis, two cases developed VT and one had VF as reperfusion arrhythmia. Both were managed with electrical cardio version followed by amiodarone infusion for 12–24 h. All safety precautions as per COVID protocol were followed in the view of safety of patient as well as health-care providers. Inotropes (noradrenaline infusions) were used for two cardiogenic shock cases. Intra-aortic balloon pump, extracorporeal membrane oxygenation, and LV assist devices were not used because of unavailability. One young male (38 years) with AWMI had AWMI in COVID ward. He was lysed and referred to private hospital for further evaluation because he

Table 9: The COVID-related conditions for the COVID ACS patients

COVID-related conditions	Status	STEMI	NSTEMI	Unstable angina
RT-PCR	+ve	29	3	15
	-ve	6	1	4
	Not done	2	1	1
CT – Chest lung involvement	Mild (<15%, CORADS – 5)	11	2	6
	Moderate (15–50%, CORADS – 5)	17	2	10
	Severe (>50%, CORADS – 5)	9	1	4
O_2 management	Room air	11	2	1
	Face mask	9	1	7
	NRM	8	2	8
	NIV – CPAP	4	-	3
	Mechanical invasive ventilator	3	-	1
	HFNO	2	-	-

RT-PCR: Reverse transcription-polymerase chain reaction, CT: Computed tomography, ACS: Acute coronary syndrome, COVID: Coronavirus disease

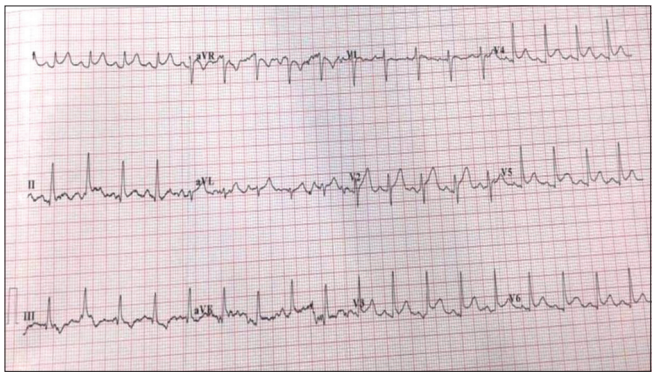
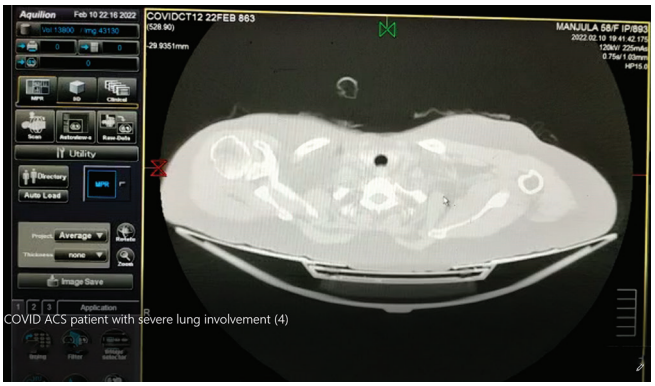


Figure 4: The ECG of COVID ACS patient with diffused ST elevation



Video 1: The CT cine video of COVID ACS patient with severe lung involvement (GGO – 90%)

wanted to go for it. We followed him; he was subjected to CT CAG and CAG. The left anterior descending artery showed 90% lesion which was stented. All other 61 cases were given only medical management. Role of bedside echo by point-of-care ultrasound[12] was useful to arrive at final diagnosis. Screening echo showed massive PE in one case and pulmonary embolism in another case. Both dealt accordingly. Both cases were having atypical angina and ST and T changes with tachycardia in the ECG. Pulmonary embolism patient was lysed with alteplase but he succumbed due to >95% lung involvement. Massive PE case survived after echo-guided pericardiocentesis. Three cases were COVID tested positive and one patient had severe lung involvement [Video 1]. Many (47 out of 62) were tested RT-PCR positive. Fourteen out of 62 cases had severe lung involvement. One case needed mechanical ventilation; other cases treated with continuous positive airway pressure (three cases), non-rebreather mask (eight cases), and facemask (seven cases) [Table 9].

Outcome

It is observed that there is little difference in in-hospital mortality between COVID ACS cases and non-COVID ACS cases. Out of 62 ACS cases with COVID-19, in-hospital mortality rate was 22.6%.[13] Out of 1541 ACS cases without COVID, in-hospital mortality rate was 15.4%. Half of them had DM as risk factor. In-hospital mortality rate is higher in males than females.[14] STEMI group shows higher deaths between ages 60 and 69 years. Most deaths (57%) occurred within 24 h.

Limitations of Study

In-hospital mortality may not represent true long-term outcomes that were not assessed in our study. Details on guideline directed medical therapy, duration of hospital stay, and risk factors for mortality such as arrhythmias have not been included in the study. Our study may not reflect the true population incidence of MI due to transport issues and referral bias.

CONCLUSION

In COVID pandemic, ACS patients seek medical attention and care after a period of delay due to fear of acquiring infection[15] and other social causes. There was dilemma in diagnosing[16] and classifying them as STEMI, NSTEMI, and unstable angina because of many mimickers. Even after diagnosis of ACS with COVID, there is difficulty in managing them with invasive strategy. There is high incidence

of unstable angina in COVID ACS when comparing to non-COVID ACS. In our Study, there were no female ACS patients with COVID-19 below 50 years.Thrombolytic therapy alone saved 85% of COVID ACS cases.[17] The in-hospital mortality in COVID ACS is not only due to MI[18] but also due to severe COVID pneumonia. The in-hospital mortality in COVID ACS treated with mild-to-moderate COVID pneumonia is comparable with non-COVID ACS treated with invasive strategy.

ETHICAL APPROVAL

This study was approved by the Institutional Ethics Committee.

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