

Early Invasive Ventilation and Early Extubation to Niv- The Management of Covid-19 Patient with Severe Acute Respiratory Failure

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Abstract

Covid-19 pandemic since its inception has reshaped many healthcare practices. One of the most severely affected speciality is intensive care and multiple strategies have been applied to deal with the severe covid-19 related pneumonia. This case report focuses on early invasive ventilation strategy versus delayed invasive ventilation strategies and early extubation and subsequent post extubation support with Non-invasive ventilation strategies.

Keywords: COVID-19; Respiratory failure; ARDS; SARS-COV₂.

Introduction

The world is facing the novel Corona Virus related COVID-19 disease since December 2019. It has been declared as global pandemic since March 11, 2020 [1]. The Corona Virus is a single- stranded RNA virus named as SARS-CoV 2.

As of December 11, 2020, almost one year since the first case reported in Wuhan China, the world has seen more than 68 million confirmed cases and more than 1.5 million deaths reported to World Health

Organization (WHO). Most of the people (approximately 80%) infected with COVID-19 require no special treatment or hospitalization but some may develop serious illness [2]. Around 1 in 5 people infected with virus acquire serious illness and develop respiratory distress of variable intensity requiring hospitalization.

Among those admitted to the hospital 20-40% require critical care and deaths among those requiring critical care is around 40% globally.

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Since the onset of disease, the world has seen huge number of studies, case reports, research and data being published regarding prevention, diagnosis, and management of the COVID-19 patient. In this case report, the key areas of management of a COVID-19 patient complicated by Acute Respiratory distress requiring invasive ventilation are outlined.

Background

Covid-19 symptoms usually start after 5 days of infection which includes fever, dry cough, body aches and loss of taste and smell. After 12 days of infection some people develop breathlessness and more frequent cough. This is the stage where most people go to hospitals. It has been seen that People who are immunocompromised due to some chronic medical conditions have higher rates of medical admission with oxygen requirement than healthy population. Researches and surveys all over the world has also reported higher ICU admissions and higher mortalities for these patients.

Some patients do recover after Mechanical ventilation but develop long Covid-19 symptoms afterwards. COV-SARS₂ causes primarily a mix of respiratory, gastrointestinal, cardiovascular, and neurological and thromboembolic disease with variable degree of involvement of different organ systems.

It was previously considered as mainly a respiratory system virus causing pneumonia of variable severity and acute respiratory distress syndrome [3]. It still mainly remains a respiratory system virus as bilateral pulmonary infiltrates with decreased level of

oxygenation in the blood are a frequent finding in most of the moderate to severe intensity cases. By the end of 2020, FDA approved many treatment strategies including monoclonal antibodies, steroids, and convalescent plasma for critical COV-SARS 2 patients. This case report includes summary of patient who recovered without any plasma and tocilizumab administration just by following good intensive care pharmacological and mechanical strategies.

Case report

A 39-year-old male was admitted with the history of fever for last 10 days and shortness of breath for last 2 days. The patient was previously not known to have any illness and was started on symptomatic treatment initially by a local general practitioner, but condition did not improve. Patient was advised by GP to get SARS CoV-2 rPCR done and it came out to be positive on 11th of May 2020. Patient got admitted on 12th of May to the hospital and was maintaining saturation up to 93% on 5-6 litres of oxygen via facemask.

On examination, patient was found to be overweight having BMI of 29. Patient was tachycardiac with a heart rate of 105/min, temperature of 37.2 degree Celsius and respiratory rate of 24/min while rest of the examination was unremarkable. Patient inflammatory markers at admission were as follows: CRP-65, serum ferritin levels-61 and D-Dimer levels-190. Patient was started on methylprednisolone 40 mg twice daily (BiD) orally or via NGT, enoxaparin 80 mg BiD subcutaneously and azithromycin 500 mg OD (as initial trials showed its immunomodulation role in mitigating

excessive inflammation and benefit in tissue repair).

Orally along with symptomatic treatment for fever and cough. Chest X-ray at presentation showed bilateral patchy opacities and HRCT showed bilateral ground glass haziness in lower and middle zones most dense posteriorly and slightly patchy anteriorly [3].

Three days after admission, patient respiratory compromise further worsened and patient oxygen requirement increased drastically and was maintaining saturation only up to 86% at 15 L/min via non-rebreather face mask. Patient was immediately shifted to ICU for further management. Patient eventually developed hypoxemic respiratory failure (type-1) which was confirmed on arterial blood gas report. Patient Arterial blood gas showed PH of 7.3, PO₂ of 6.5 and PCO₂ of 5.5. Patient was initially started on CPAP but was unable to tolerate it for long and was desaturating to 82%. Patient was given CPAP trial for 6-8 hours.

Eventually patient was electively intubated and ventilated on pressure control mode with PEEP of 8 cm of H₂O and pressure support of 20 cm of H₂O with a respiratory rate of 15/min. Patient was kept sedated and paralyzed initially for the first 3 days. Patient remained hemodynamically stable till 3rd day of ICU admission.

Ventilatory requirements, ECG and CXR changes remain grossly unchanged in the initial 3 days. On day 4 of patient ICU admission, patient developed sinus bradycardia and sudden episode of desaturation which responded to maximum ventilatory support.

Azithromycin was put on hold in response to bradycardia and borderline QT-interval [4]. Patient was continued on therapeutic thromboprophylaxis along with pneumatic compression devices.

PaO₂:FiO₂ showed mild ARDS with a value less than 300 mm of Hg [5]. Patient persistently started spiking fever from day 4 onwards of ventilation. Blood and sputum cultures were sent and patient was started on Tanzocin 4.5g 6 Hourly as empirical therapy for VAP or septicemia [6]. Patient responded well to the change in treatment and fever also settled in next 24 hours. From ventilatory day 6 onwards patient was regularly given sedation breaks and weaning trials.

FiO₂ was gradually tapered down to 0.5 and PEEP to 5 cm of H₂O. On day 7, patient was given spontaneous breathing trial with PS, which patient tolerated quite well. Patient developed no fever in last 24-48 hours and patient inflammatory markers showed improvement. On the basis of clinical and laboratory numbers patient was eventually extubated on day 8 of IPPV. Patient Pre-Extubation ABGs showed PH of 7.42, PO₂ of 8.5 and PCO₂ of 4.5 After extubation.

Patient remained on intermittent CPAP especially in the night times with high flow oxygen with FiO₂ of 60% for initial few days post-extubation. Patient also developed ICU delirium during the patient stay in the ICU which was treated with psychotherapy and anxiolytics.

Oxygen requirement kept on decreasing as the patient was gradually mobilized out of bed. Chest physiotherapy and incentive spirometry was continued every 2 hourly [7].

Patient also required extended physiotherapy to regain muscle strength [8]. Patient was eventually discharged from the hospital on 17th day of patient admission. Patient

remained on follow-up with Internal Medicine and Pulmonology for the initial 3 months and continued outpatient chest and limbs physiotherapy.



Figure 1: Post-intubation day 1 CXR.

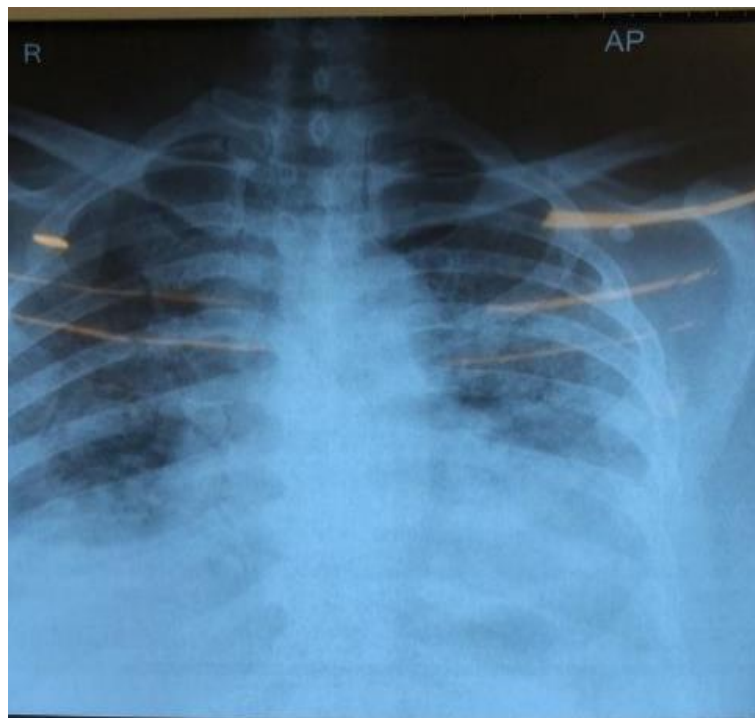


Figure 2: Post-extubation day 2 CXR.

Summary of the Laboratory Values								
LABS	Pre-Vent	Vent day 1	Vent day 2	Vent Day 5	Vent Day 6	Pre-Extubation	Post-Extubation Day 1	Pre-Discharge
PO ₂	63	95	89	93	-	95 (on 60% FiO ₂ , PEEP of 8)	85	89 mmHg
LAC	-	2.7	1.7	-	1.5	-	-	-
Cr	0.7	0.9	0.9	-	-	-	0.8	0.6 mg/dL
Na	-	141	142	-	-	-	143	135 mEq/L
K	-	3.4	3.8	-	-	-	4	4.9 mmol/L
ALB	-	2.6	-	-	-	-	-	-
ALT	-	31	-	-	-	-	-	-
AST	-	20	-	-	-	-	-	-
Ferritin	61	-	80	300	-	-	-	-
PO ₂ /FIO ₂	170	210	350	250	260	-	-	-
PCT (ng/ml)			0.08	1.5		-	0.05	-
CRP	38	170	53.8	30	20	-	10	6.7
D-DIMER (ng/)	190	-	-	600	-	-	200	-
WBC	-	11.3	10.4	15	13	-	12	10

Table 1: Summary of the laboratory values.

Discussion

It causes primarily a mix of respiratory, gastrointestinal, cardiovascular, and neurological and thromboembolic disease with variable degree of involvement of different organ systems. It was previously considered as mainly a respiratory system virus causing pneumonia of variable severity and acute respiratory distress syndrome [9]. It still mainly remains a respiratory system virus as bilateral pulmonary infiltrates with decreased level of oxygenation in the blood are a frequent finding in most of the moderate to severe intensity cases. Wearing masks, practice of social distancing and vaccination has significantly reduced the infection rate and its severity. Most of the world now is entering its 3rd or 4th wave with different variants. Dealing with severe respiratory

disease still largely remains a challenge for most of the intensive care units.

In this case report early invasive ventilation strategy is emphasized in comparison to the NIV strategies in select young individuals who may benefit from this therapy along with steroids and other ICU supportive therapy. Also, early extubation to NIV despite the high FiO₂ requirements and relatively higher PEEP values might be acceptable to avoid prolonged invasive ventilation and its complication. This strategy though might not be suitable for all kind of patients, but it can be a useful strategy in select patients to avoid the initial harmful effects of prolonged NIV like pneumothorax and pneumo-mediastinum which can also make invasive ventilation even more challenging in already difficult patients.

Conclusion

1. Patients have acute respiratory insult secondary to COV-SARS-2 in hospitals might benefit from early intubation and subsequent early extubation trial [10].
2. Early intubation may prevent lung injury in patients who breath spontaneously and have high respiratory drive. Early invasive mechanical ventilation also acts as means to reduce aerosolization of virus in Covid-19 patients, as would happen alternately by high flow oxygenation or CPAP allowing air leaks. Contrary some studies and

clinical data suggest that ventilation in Covid-19 patients also put them on risk of ventilator associated pneumonia, lung injury, complications linked to sedatives and paralytics and barotrauma.

3. Good sedation and ventilation in initial few days can achieve good Ventilation/Perfusion ratios and thereby prevent/improve early ARDS [11].
4. Chest Physiotherapy, Posture Changing and suctioning aids in alveolar recruitment along with preventing mucous plugging and these strategies altogether enhances early weaning success.

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