



Severe COVID-19 Case with HRCT Score of 25/25: A Survival Story

Param Shah¹, Satish Mahajan^{1*}, Sunil Kumar¹, Sourya Acharya¹, Mona Vohra², Charan Bagga¹ and Dhruv Talwar¹

¹Department of Medicine, Jawaharlal Nehru Medical College, Datta Meghe Institute of Medical Sciences (Deemed to be University), Sawangi (Meghe), Wardha-442001, Maharashtra, India.

²Department of Respiratory Medicine, Jawaharlal Nehru Medical College, Datta Meghe Institute of Medical Sciences (Deemed to be University), Sawangi (Meghe), Wardha-442001, Maharashtra, India.

Authors' contributions

This work was carried out in collaboration among all authors. All authors equally participated in the literature search, compilation of data and writing a case report. All authors read and approved the final manuscript.

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Case Study

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ABSTRACT

With the rapid spread of severe acute respiratory syndrome SARS-CoV2, the incidence of the disease is increasing each day with a higher infectivity rate and unpredictable morbidity and mortality. The second wave in India has higher morbidity and mortality compared to the previous wave of SARS-Cov2. With the increasing trend of screening hypoxic patients with high resolution computed tomography (HRCT) of thorax, treatment modalities, and investigational approaches have changed drastically. Here we report a case of a 31-year-old female who presented to a tertiary care centre in the rural area with a history of fever five days back and a history of breathlessness of 3 days duration and dry cough of 3 days duration. She was hypoxic on admission and later on diagnosed with SARS-Cov2 infection. HRCT thorax revealed a CT severity

score of 25/25. She was treated according to standard treatment protocols. The patient was discharged after 46 days with normal oxygen saturation on room air. The aim of this case study is to emphasize the wholesome approach in the management of COVID-19.

Keywords: SARS Cov-2; ARDS; HRCT thorax; cytokine storm; coagulopathy.

ABBREVIATIONS

HRCT	: High resolution computerized tomography
MMRC	: Modified medical research council
ARDS	: Acute respiratory distress syndrome
SARS-CoV2	: Sever acute respiratory syndrome-Coronavirus-2
COVID-19	: Coronavirus disease-2019
CORAD	: Coronavirus disease-2019 reporting and data system
TLC	: Total leucocyte count
N:L	: Neutrophil to lymphocyte ratio
CRP	: C-reactive protein
LDH	: Lactic dehydrogenase
SGOT	: Serum glutamic oxaloacetic transaminase
SGPT	: Serum glutamic pyruvic transaminase
RT-PCR	: Reverse transcriptase- polymerase chain reaction
Spo2	: Saturation of Oxygen
BiPAP	: Bilevel positive airway pressure
FiO2	: Fraction of inspired oxygen
CT	: Cycle threshold
IL-6	: Interleukin-6

1. INTRODUCTION

The SARS-Cov2 infection causes acute respiratory failure secondary to direct respiratory tract injury to terminal bronchioles and alveoli. Hypoxia is also contributed by underlying hyper inflammation and coagulopathy in the pulmonary vasculature [1]. HRCT thorax has become the usual approach modality to any patient with confirmed SARS-Cov2 presenting with hypoxia. HRCT thorax aids in detecting lesions qualitatively in terms of 'CORAD scoring' and quantification of lesions based on the involvement of different lobes in terms of 'CT SEVERITY SCORE'. As various protocols stating no role of HRCT thorax in the initial management strategy, it has a role in predicting the further course of illness and initiating necessary treatment in the early stages [2]. The aim of this case study is to emphasize the wholesome approach in the management of COVID-19.

2. CASE HISTORY

2.1 Patient Information

A 31-year-old female presented to us with a history of a single episode of fever five days back, breathlessness for three days, dry cough for three days. The breathlessness was sudden in onset and gradually progressed from MMRC grade II to MMRC grade IV over three days. The cough was non-productive. The patient had no history of bronchial asthma or interstitial lung disease or valvular heart disease.

2.2 Clinical Findings

On admission, the patient was in respiratory distress and was using accessory muscles of respiration with a respiratory rate of 42/min with Pulse rate: 116/min (regular), Blood pressure: 110/70 mmHg, Oxygen saturation on room air: 67%.

Findings of inspection and palpation of systemic examination revealed normal jugular venous pressure, bilateral equal chest rise, no tracheal deviation on palpation of the trachea, dullness over an infra-scapular and infra-axillary area, intact superficial cardiac dullness. The patient could not be auscultated due to the use of Personal protective equipment.

2.3 Diagnostic Assessment

The patient's laboratory assessment showed TLC: 2800 cells/mm³ showing neutrophilic leucocytosis, N:L ratio was 8.5, CRP: 68.9, D-dimer: 2080 ng/ml, Ferritin: 866 microgram/L, LDH: 1000 U/L, SGOT: 120 U/L, SGPT: 92 U/L, Procalcitonin: 0.2 ng/ml, Interleukin-6: 114 pg/ml.

The patient's serial inflammatory markers are summarised in a Table 1.

Fig. 1 to Fig. 3 showing HRCT Thorax sections at (Fig. 1) trachea (Fig. 2) carina and (Fig. 3) heart level showing extensive ground-glass opacities including subpleural areas with diffuse organizing pneumonia with interlobular septal thickening and pleural lesions.

Table 1. Serial inflammatory markers

	CRP (mg/L)	D-dimer (ng/ml)	Ferritin (microgram/L)	LDH (U/L)	IL-6 (pg/ml)
Normal values	0.8-1.0	0-500	10-250	140-280	0-16.4
Day 1	68.9	2080	866	1000	114
Day 3	178	4580	1285	1456	787
Day 6	35	940	784	940	72

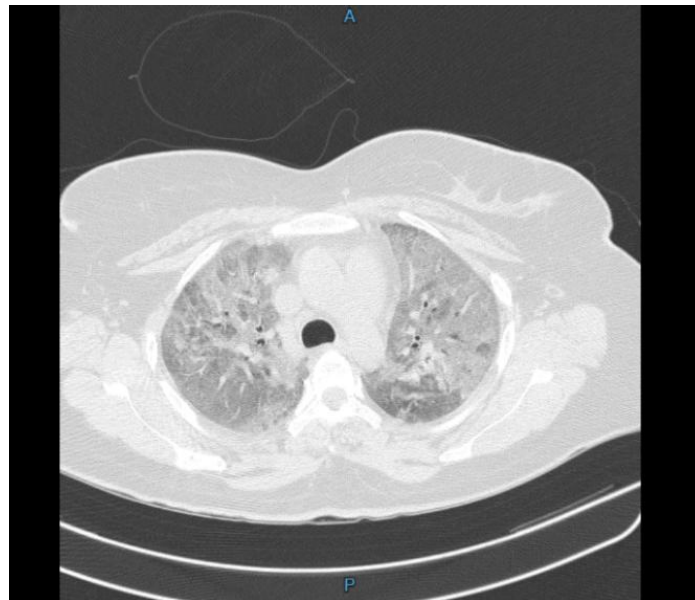


Fig. 1. HRCT Thorax section at the level of trachea showing extensive ground-glass opacities including subpleural areas with diffuse organizing pneumonia with interlobular septal thickening and pleural lesions



Fig. 2. HRCT Thorax section at the level of carina showing extensive ground-glass opacities including subpleural areas with diffuse organizing pneumonia with interlobular septal thickening and pleural lesions



Fig. 3. HRCT Thorax section at the level of heart showing extensive ground-glass opacities including subpleural areas with diffuse organizing pneumonia with interlobular septal thickening and pleural lesions

2.4 Therapeutic Intervention

The patient was initially given high flow oxygen through a Hudson mask on which the patient could maintain 78% of Spo₂. RT-PCR for SARS-CoV2 was sent. Later on, she was put on non-invasive BiPAP ventilation with Fio₂ of 100%, on which she was able to maintain 87% of Spo₂. Later, she came out to be reactive to SARS-CoV2 with a COVID-19 eGene CT value of 18.

The patient was started on Injectable antiviral Remdesivir. It was given for five days, IV steroids in the form of methylprednisolone for 12 days, subcutaneous low molecular weight heparin for 15 days in its therapeutic dose since the patient had no contraindications for therapeutic use anticoagulation. The patient was also started on an inhalational steroid in the form of nebulization of budesonide. The patient was intermittently given prone ventilation. Human regular insulin was given according to blood sugar levels.

After 48 hours of admission, inflammatory markers were repeated which showed a rising trend. Given her clinical deterioration and rise in markers, Injection Tocilizumab 400mg was given to the patient, followed by a repeat dose after 12 hours.

On Day 13, the patient's TLC was raised to 28,300 cells/mm³, with an N:L ratio of 6.8. Procalcitonin levels increased to 28.6ng/ml. Her blood pressure fell to 78mmHg and she was started on inotropic supports in the form of noradrenaline and vasopressin. Later, the patient was weaned off from inotropic supports.

Later on, the patient showed clinical improvement in the form of a rise in Spo₂ and declining respiratory distress. The patient was put on High flow oxygen through Hudson mask and the patient maintained 92% Spo₂.

The patient was started on the antifibrotic agent Pirfenidone. At this stage, the treatment regimen was switched to oral anticoagulants and pulmonary rehabilitation was initiated in the form of chest physiotherapy and incentive spirometry.

2.5 Outcome

On discharge, the patient had significant improvement in respiratory distress. She was able to maintain Spo₂ of 95% on room air. The patient was advised for follow-up HRCT after three months.

2.6 Patient Perspective

At first, when I was brought to the emergency medicine department, checking upon my oxygen saturation frightened me. The visuals in the emergency medicine department at the peak of the COVID-19 outbreak seemed mortal to me. It seemed almost impossible to get out of the noisy ICU sounds. BiPAP ventilation provoked my claustrophobia. However, with the great care and empathy of the hospital staff, I battled this war.

3. DISCUSSION

Major causes leading to mortality in severe SARS-CoV2 cases are ARDS and coagulopathy, which is a consequence of uncontrolled inflammatory processes [3]. When SARS-CoV2 invades airway epithelium, it does not trigger the release of interferons which constitutes the primary defence to arrest viral replication. In these cases, the infected epithelia secrete IL-6 and pro-inflammatory cytokines, which pool monocytes and cytotoxic T cells to the infection site to recognize and to kill the infected cells [4].

SARS-CoV-2 infection is cleared in normal subjects through this mechanism, the magnitude of inflammation declines, and patients recover. In severe cases, however, increased secretion of IL-6 and other pro-inflammatory cytokines summon T cell aggregation and cause T cell functional exhaustion [5].

In addition, the cytokine storm also destroys vascular endothelium, hemostasis, and the activation of coagulation, triggering coagulopathy in these patients. It has been well-established that COVID-19-induced cytokine storm has the main role in COVID-19 related mortality [6].

While there have not been any approved modalities for the COVID-19-induced cytokine storm, various treatments directed at the preventing and suppressing cytokine storms have been proposed. Glucocorticoids have been proved effective anti-inflammatory therapy and were widely implemented during the epidemics of SARS and MERS. The first week of covid 19 infection signifies viral replication where administering glucocorticoids are a bane than a boon [7]. Their role is significant in the second week of the disease or particularly at the onset of dyspnoea to prevent the "cytokine storm" progression [8].

Though the drug Remdesivir is an antiviral agent, it is not approved by the FDA. The "Emergency Use Authorization" (EUA) for moderate to severe cases is being endorsed. Remdesivir is effective in reducing hospital stay—but it is not a cure-all. Studies suggest its effectiveness within the first ten days of onset of illness. This case of severe covid 19 infections with the CT score of 25 was an all or none situation for us [9].

IL-6 being the chief contributory cytokine in COVID-19 induced cytokine storm, aiming blockade of IL-6 receptor is essential. Tocilizumab is a competitive inhibitor of both the membrane-bound and soluble IL-6 receptor. Based on our case, we speculate that early Tocilizumab administration in patients who develop sudden clinical deterioration 7–10 days from the initial onset of symptoms will reduce fatality among severe COVID-19 patients with a rapid disease trajectory [10]. It also reduces the risk of requiring invasive ventilation and offers a chance of survival to people who are unfit for escalation or have refused to be ventilated [11]. Inflammatory markers mainly IL-6, CRP, and ferritin, before and after administration of tocilizumab have been studied to assess the efficacy of tocilizumab [12].

We also carried a wholesome approach where we emphasized very much the supportive and nutritional aspect of the treatment. We focused on prone ventilation as an adjunct to improve ventilation and blood supply to the lungs, adequate fluid management, and prevention of post covid complications by giving long-term anticoagulant, anti-fibrotic agent [13].

Our case seems to denote that a highly sensitive imaging modality like CT, although not as specific, might be beneficial to speed up diagnostic and therapeutic workup. CT damage is highly correlated with various disease parameters, including clinical staging and laboratory parameters [11].

Probable limitations of our study are in terms of variables like age, co-morbidities, the point of initiation of treatment in the disease course. These variables may differ in case-to-case basis, translating into favourable to lethal outcomes.

In conclusion, raised acute phase reactants, ARDS, lymphocytopenia, and elevated CT score are chief predictors of mortality in severe COVID-19 patients. For patients who contracted severe COVID-19, a follow-up CT scan after three months might be important for assessing the outcome in patients with severe COVID-19. Baseline HRCT scan of the thorax is directly proportional to morbidity and mortality in COVID-19, but in our case despite the CT severity score of 25/25, we were able to discharge the patient with normal oxygen saturation on room air [12].

4. CONCLUSION

This case to us was quite discouraging at the presentation and her disease trajectory was also steep at first. It seemed unattainable to treat such a morbid presentation. In addition to her clinical presentation, HRCT thorax also revealed a grave prognosis. Despite her rapid deterioration and severe CT score, the wholesome and aggressive approach in terms of early use of anti-inflammatory measures and extended use of antivirals proved vital to the patient's recovery. Our case also gravitated to the use of antifibrotics and chest physiotherapy to prevent long-term morbidity related to COVID-19.

DISCLAIMER

The products used for this research are commonly and predominantly use products in our

area of research and country. There is absolutely no conflict of interest between the authors and producers of the products because we do not intend to use these products as an avenue for any litigation but for the advancement of knowledge. Also, the research was not funded by the producing company rather it was funded by personal efforts of the authors.

CONSENT

The patient was informed about the case report and why her case would be an invaluable addition to the available medical information related to her condition. She gave informed consent to have this case report written up and published.

ETHICAL APPROVAL

As per international standard or university standard written ethical approval has been collected and preserved by the author(s).

COMPETING INTERESTS

Authors have declared that no competing interests exist.

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