CASE REPORT

High-flow Oxygen Therapy via Tracheostomy to Liberate COVID-19-induced ARDS from Invasive Ventilation: A Case Series

Sonali Vadi[1], Sourabh Phadtare[2], Kiran Shetty[3]

Abstract

Lung involvement with differing phenotypes characterizes COVID-19-induced acute respiratory distress syndrome (CARDS). The liberation of these patients from mechanical ventilation has been challenging. Excessive stress and strain following increased respiratory efforts spiral their vulnerable lung tissue into ventilator-induced lung injury vortex. The use of high-flow oxygen therapy via tracheostomy ($HFOT_{Tracheal}$)eases weaning process. As a safe option for both the patient and the healthcare workers, $HFOT_{Tracheal}$ was successfully employed to wean two CARDS patients from the mechanical ventilator.

Keywords: COVID-19-associated ARDS, High-flow oxygen therapy via tracheostomy, Patient–ventilator interaction, Ventilator-induced lung injury, Weaning from mechanical ventilation.

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Introduction

COVID-19 patient with acute respiratory distress syndrome (CARDS) are on a spectrum of a characteristic high compliance lung initially, with lower lung weight on CT scan, the type L,[1] or phenotype 1[2] who may evolve to a type H[1], or phenotype 3[2] characterized by low compliance, extensive consolidations on CT scan. Although severely hypoxemic, their lung mechanics are generally preserved. Interactions between ventilator and the injured lung impact lung injury and its repair. Consequently, the shrunken and vulnerable baby lung incur damage, ventilator-induced lung injury (VILI). Tidal cycling, cycling frequency, and airway pressures influence VILI. Each phenotype benefits from personalized ventilator and weaning strategies.

Lung protective ventilation may not necessarily be "protective" for those breathing spontaneously on mechanical ventilator. Tachypnea and in-coordinated respiratory efforts lead to a nonhomogeneous distribution of the stress and strain in the healthy regions of the lung. This is patient self-inflicted lung injury (P-SILI).[3] P-SILI is associated with high respiratory rates and irregular tidal volumes. Spontaneous efforts may lead to heart-lung imbalances. These patients benefit from customized weaning.

High-flow oxygen therapy (HFOT) has been studied in tracheostomized ($HFOT_{Tracheal}$) patients who are at high risk of weaning failure.[4] Reduced inspiratory effort with $HFOT_{Tracheal}$ facilitated weaning from prolonged mechanical ventilation in two patients with restrictive pulmonary function.[5] Herein is discussed the use of $HFOT_{Tracheal}$ to successfully wean off two patients with CARDS.

Case Description

Patient 1

A 50-year-old male, hypertensive with type II diabetes mellitus, was transferred to the COVID-19 unit on 15 L oxygen via the nonrebreather mask (7.45/31.9/55.8/23). Five days later following desaturation, he was switched to high-flow oxygen therapy via nasal cannula ($HFOT_{Nasal}$): 60 L flow rate, 0.65. Oxygen requirements on $HFOT_{Nasal}$ progressively increased and on day 10 of COVID-19 unit stay he had to be invasively ventilated. HRCT chest (noncontrast) revealed >75% of lung involvement (Fig. 1). Methylprednisolone, Remdesivir, Tocilizumab, and Enoxaparin were administered at the outset. Chest X-ray revealed a progressive increase in pulmonary infiltrates. Fivedays later he was tracheostomized. The following day he was transferred to the regular ICU.He required 0.6/+5(pH 7.47/$PaCO_2$47.5/$PaO_2$73.3/HCO_3^-35.9). Weaning attempts were commenced 72 hours later (Table 1). His pulse oximeter saturations would drop to <90% with a reduction in FiO_2 to <0.6. Ventilatory need was associated with anxiety and fear leading to patient–ventilator dyssynchrony. This required sedation.Opioid useresulted in bowel dilatation mandating its stoppage. Dexmedetomidine use was associated with consequent bradycardia requiring discontinuation. Subsequently, small dose of Midazolam infusion was prescribed. These prolonged his time on the ventilator.

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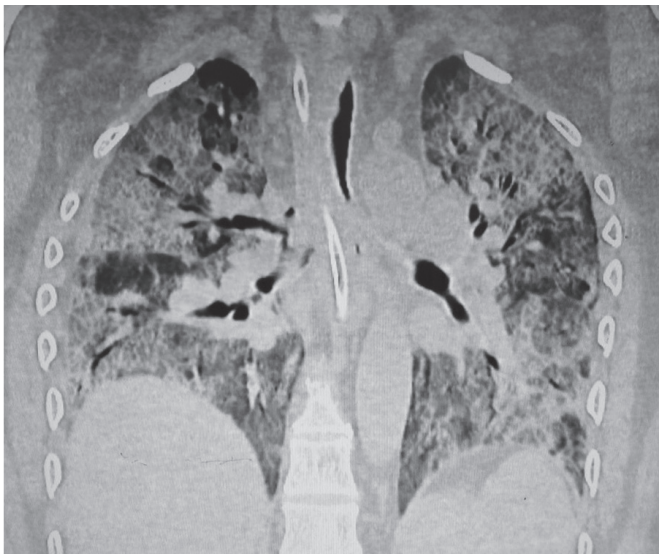

Fig. 1: (Patient 1) HRCT chest (noncontrast)

Table 1: Patient 1—ventilator parameters and high-flow oxygen therapy via tracheostomy

Patient1: ventilator weaning and respiratory parameters						
ICU day	*Cumulative duration of T-piece trials*	*Maximum FiO_2, PEEP*	*Maximum respiratory rate (breaths/min)*	*SpO_2 (min–max)*	*Arterial blood gas: pH/$PaCO_2$ (mm Hg)/PaO_2 (mm Hg)/ HCO_3 (mEq/L)*	*P/F ratio*
ICU day 1	None	0.6, +6	32	93–100%	7.35/67/79.2/35.7	132
ICU day 3	None	0.4, +5	36	93–100%	7.47/44.1/66.2/32.9	165
ICU day 4	7 hours	0.4, +5	40	88–99%	7.48/46/104.5/35.1	361
ICU day 5	9 hours	0.6, +5	34	88–98%	7.50/46.7/55.2/37	92
ICU day 6	2 hours	0.6, +5	31	90–99%	7.31/70.2/64.2/36.9	107
ICU day 7	10 hours	0.6, +5	38	86–94%	7.47/47.7/45.5/35.6	75.8
ICU day 9	6 hours	0.6, +6	32	90–100%	7.50/38.2/53.3/30.4	88.8
ICU day 11	9 hours	0.6, +6	32	90–100%	—	—
$HFOT_{Tracheal}$						
	HFOT Tracheal: FiO2	*HFOT Tracheal: Flow (L/min)*	*Maximum respiratory rate (breaths/min)*	*SpO2*	*Arterial blood gas: pH/PaCO2 (mm Hg)/PaO2 (mm Hg)/HCO3 (mEq/L)*	*P/F ratio*
ICU day 13	0.4	50 L	38	94–99%	—	—
ICU day 14	0.3	40 L	36	90–98%	—	—
ICU day 15	0.3	30 L	39	92–100%	—	—
ICU day 16	0.3	25 L	30	93–99%	7.49/36.4/64.7/28.4	215
ICU day 17	$HFOT_{Tracheal}$ switched to T-piece with 4 L oxygen		24	94–100%	—	—

High-flow oxygen therapy via tracheostomy ($HFOT_{Tracheal}$), inspired fraction of oxygen (FiO_2), positive end-expiratory pressure (PEEP), PaO_2/FiO_2 (P/F) ratio

On ICU day 13, he was switched from the ventilator to $HFOT_{Tracheal}$ (Table 1). AIRVO™ 2 system (Fisher and Paykel Healthcare) with an HFO interface, Optiflow™(Fisher and Paykel Healthcare) for tracheostomy was employed. $HFOT_{Tracheal}$ was weaned off in 4 days, FiO_2 first followed by the flow. T-piece was weaned off over next 5 days with SpO_2 ranging from 97 to 98% on room air. Respiratory physiotherapy was continued daily throughout the weaning efforts.

Patient 2

A 67-year-old male was transferred to the COVID unit requiring 15 L oxygen via nonrebreather mask. This had to be escalated to high-flow oxygen via nasal cannula at 0.9/60 L in less than 24 hours and invasive ventilation soon thereafter (Table 2). Methylprednisolone, Remdesivir, Tocilizumab, and Enoxaparin were administered from the day of admission given the severity of pneumonia.

Table 2: Patient 2—ventilator parameters and high-flow oxygen therapy via tracheostomy

Patient 2: ventilator settings and respiratory parameters					
	Maximum FiO2, PEEP	*Maximum respiratory rate (breaths/min)*	*SpO2 (min–max)*	*Arterial blood gas: pH/ PaCO2 (mm Hg)/PaO2 (mm Hg)/HCO3 (mEq/L)*	*P/F ratio*
COVID ICU day 3	1.0, +10	22	93–99%	7.39/49/77.2/31	77
COVID ICU day 5	0.6, +7	20	91–97%	7.46/42/77.8/31.3	129
COVID ICU day 7	0.75, +6	20	93–97%	7.42/45.4/55.6/30.7	74
COVID ICU day 9	0.8, +8	20	90–98%	7.41/54.9/56.2/35.7	70
COVID ICU day 11	0.7, +8	20	93–98%	7.45/53.5/70.9/38.7	101
COVID ICU day 13	0.8, +8	20	90–97%	7.48/54/69.8/41.4	87
COVID ICU day 15	1.0, +12	20	88–95%	7.48/46.7/63.5/36	63
COVID ICU day 17	0.6, +10	24	92–98%	7.51/43/55.9/35	93
COVID ICU day 19	0.6, +10	24	90–95%	7.45/44/75.2/31.3	125
$HFOT_{Tracheal}$					
	HFOT Tracheal: FiO2	*HFOT Tracheal: Flow (L/min)*	*Maximum respiratory rate (breaths/min)*	*SpO2*	*Arterial blood gas: pH/ PaCO2 (mm Hg)/PaO2 (mm Hg)/HCO3 (mEq/L)*	*P/F ratio*
ICU day 20	0.6	60 L	23	90–95%	—	—
ICU day 22	0.55	60 L	32	88–100%	7.47/37.3/71.4/27	130
ICU day 24	0.7	60 L	25	89–100%	—	—
ICU day 26	0.50	60 L	29	89–98%	7.51/28.6/69.8/23.2	140
ICU day 30	0.45	60 L	26	90–95%	—	—
ICU day 32	HFOTracheal switched to T-piece with 6 L oxygen		29	91–98%	—	—

High-flow oxygen therapy via tracheostomy ($HFOT_{Tracheal}$), inspired fraction of oxygen (FiO_2), positive end-expiratory pressure (PEEP), PaO_2/FiO_2 (P/F) ratio

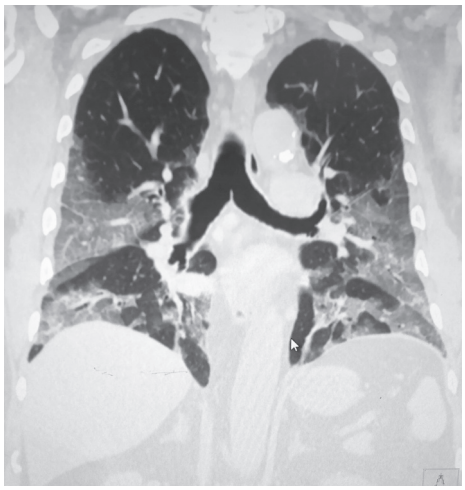

Fig. 2: (Patient 2) HRCT chest (noncontrast)

Tracheostomized, he was transferred out to the regular ICU on day 20. The following day, he was switched to $HFOT_{Tracheal}$ and weaned off the same over the next 5 days (Table 2), FiO_2 first followed by the flow. His lung involvement on HRCT chest was at 60% (Fig. 2).

Discussion

Patient 1

Temporal chest X-ray findings worsened in parallel to his clinical deterioration prior to his requiring intubation, reflecting pulmonary progression of primary COVID-19 infection. Ventilator weaning was a slow and laborious process for the patient. This patient was hypoxic. He tired out easily during T-piece trials with ensuing tachypnea and dyspnea. Weaning attempts were associated with patient–ventilator dyssynchrony.Re-ventilation took time to abate these respiratory derangements. Following COVID-19 pulmonary infection, this patient developed restrictive pulmonary function as seen by fibrosis on his HRCT chest. Exertion of respiratory muscles with an excess negative force may have contributed to SILI of his CARDS lung in patient 1. Sedation with its subsequent side-effectsprolonged his time on the ventilator. On switching to $HFOT_{Tracheal}$, his oxygen requirements were lower (Table 2). Subjective dyspnea lessened and soon abated.Patient comfort improved.

Patient 2

Bearing in mind the experience of the previous patient, he was switched to $HFOT_{Tracheal}$ after a day of weaning attempt that was rapidly weaned off. Subjective dyspnea on T-piece during mobilization was managed temporarily with $HFOT_{Tracheal}$.

Risk factors of prolonged weaning are VILI, the need for sedation, deconditioning, and cost. Pulmonary physiology is dynamic.Irrespective of the ventilator mode, the respiratory rate, local inflammation caused by volutrauma,[6] repeated exposures to tidalcycles[7] coupled with their duration, and stresses within vessels[1] contribute to VILI.[8] Patient–ventilator dyssynchrony associated with irregular and dynamic spontaneous efforts teamed with erratic tidal volumes were contributors to VILI in this patient. Inhomogeneous involvement of the lung by the primary pathology acts as a stress raiser.[9] The hampered endogenous capacity of the COVID lung to prevent or repair this injury and contribute to VILI and its progression.These airspace stresses reinforce each other spiraling

Table 3: (Patient 2) Ventilator: oxygen supplied, and respiratory parameters

Patient 2	*Maximum FiO2, PEEP*	*Maximum respiratory rate (breaths/min)*	*SpO2 (min–max)*	*Arterial blood gas: pH/PaCO2 (mm Hg)/PaO2 (mm Hg)/HCO3 (mEq/L)*	*P/F ratio*
COVID unit day 3	1.0, +10	22	93–99%	7.39/49/77.2/31	77
COVID unit day 5	0.6, +7	20	91–97%	7.46/42/77.8/31.3	129
COVID unit day 7	0.75, +6	20	93–97%	7.42/45.4/55.6/30.7	74
COVID unit day 9	0.8, +8	20	90–98%	7.41/54.9/56.2/35.7	70
COVID unit day 11	0.7, +8	20	93–98%	7.45/53.5/70.9/38.7	101
COVID unit day 13	0.8, +8	20	90–97%	7.48/54/69.8/41.4	87
COVID unit day 15	1.0, +12	20	88–95%	7.48/46.7/63.5/36	63
COVID unit day 17	0.6, +10	24	92–98%	7.51/43/55.9/35	93
COVID unit day 19	0.6, +10	24	90–95%	7.45/44/75.2/31.3	125

Table 4: (Patient 2) High-flow oxygen therapy via tracheostomy ($HFOT_{[Tracheal]}$): settings and respiratory parameters

Patient 2	*HFOT[Tracheal]: FiO2*	*HFOT[Tracheal]: Flow (L/min)*	*Maximum respiratory rate (breaths/min)*	*SpO2*	*Arterial blood gas: pH/PaCO2 (mm Hg)/ PaO2 (mm Hg)/HCO3 (mEq/L)*	*P/F ratio*
ICU day 1	0.6	60 L	23	90–95%	—	—
ICU day 3	0.55	60 L	32	88–100%	7.47/37.3/71.4/27	130
ICU day 5	0.7	60 L	25	89–100%	—	—
ICU day 7	0.50	60 L	29	89–98%	7.51/28.6/69.8/23.2	140
ICU day 11	0.45	60 L	26	90–95%	—	—
ICU day 13	$HFO_{Tracheal}$ switched to T-piece with 6 L oxygen		29	91–98%	—	—

down into the "VILI vortex."[7] High driving pressures secondary to spontaneous efforts in severe ARDS add to lung injury,[10] P-SILI.

$HFOT_{Nasal}$ reduces inspiratory efforts, generates positive airway pressure,[11] decreases anatomical dead space, decreases minute ventilation, reduces driving transpulmonary pressure, decreases expiratory diaphragm loading, and improves oxygenation.[12] Consequential stress and strain are reduced in the injured lungs.

$HFOT_{Tracheal}$ is associated with a better matching of the delivered flow with the patient's spontaneous inspiratory flow, a positive end-expiratory pressure effect, with resultant reduced work of breathing.[4] On switching to $HFOT_{Tracheal}$, their oxygen requirements were lower. Subjective dyspnea decreased with an improvement in patient comfort. Higher flow rates likely linearly increased airway pressures,[13] with resultant recruitment of more alveoli.[14] Other physiological effects of $HFOT_{Tracheal}$ include reduced $PaCO_2$, patient–ventilator dyssynchrony, and an increase in P/F ratio. These were the likely mechanisms to the success of $HFOT_{Tracheal}$ in these patients. Ability to breathe spontaneously with $HFOT_{Tracheal}$ without any evidence of respiratory distress and without the necessity for reconnection to the ventilator for at least 48 hours was considered successful weaning (Table 3 and 4).

Other features of consequence for healthcare workers managing CARDS include inflated cuff, in-line suction catheter, and the use of filter to reduce the chances of aerosol generation with the use of $HFOT_{Tracheal}$.

Conclusion

VILI necessitates weaning from mechanical ventilation in CARDS. Worsening of lung injury as a result of spontaneous efforts during weaning leading to P-SILI requires customized weaning. Ability to wean off mechanical ventilation without any respiratory distress employing $HFOT_{Tracheal}$ merits further studies to wean CARDS from mechanical ventilation.

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