Congestion in the tube: air trapping in a ventilated patient with COVID-19 secondary to mucinous valves

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SUMMARY
A woman in her 50s was admitted to the intensive therapy unit with acute hypoxaemic respiratory failure secondary to COVID-19 pneumonitis. The patient was intubated on admission and worsening gas exchange necessitated multiple rounds of proning. She later improved, and her ventilation was switched to spontaneous mode. However, the patient started to develop air trapping with subsequent respiratory and cardiovascular compromise. Routine investigations showed no clear cause for her sudden deterioration and a suction catheter passed easily through the endotracheal tube. Bronchoscopy revealed mucinous/phlegmatic membranes had developed across the inner diameter of the endotracheal tube. This had created a one-way valve that allowed positive pressure ventilation through the tube into her lungs but only allowed a fraction of air to passively escape in expiration. This case report highlights a less commonly regarded complication associated with long-term intubation and lack of circuit humidification in the context of productive lung pathology.

BACKGROUND
Critically ill COVID-19 patients often require prolonged mechanical ventilation,1,2 which can lead to various complications and sometimes catastrophic airway emergencies, such as endotracheal tube (ETT) dislodgement or obstruction. One of the pathophysiological features seen in COVID-19 is mucus hypersecretion,3 which often leads to copious, thick secretions and mucus plugs.4–6 Frequent suctioning and appropriate circuit humidification are, thus, very important in improving ventilation and avoiding deterioration. After over a year into the pandemic, our hospital has experienced multiple airway emergencies relating to ETT obstruction with mucus plugs necessitating reintubation. With this case we want to demonstrate some key learning points.

CASE PRESENTATION
A woman in her 50s presented to hospital with a 2-day history of fever and shortness of breath. Her medical history included hypertension, type 2 diabetes mellitus and obesity. She was an ex-smoker of 20 pack-years with no evidence of emphysema or chronic obstructive pulmonary disease and obstructive sleep apnoea managed with an overnight home continuous positive airway pressure (CPAP) machine. After a day of support with CPAP on the medical ward her acute hypoxaemic respiratory failure worsened and she was intubated with an ETT of 7.5 mm internal diameter and transferred to the intensive therapy unit (ITU).

The patient had severe acute respiratory distress syndrome (ARDS), with P:F ratio <13.3 kPa and a chest radiograph showed worsening bilateral interstitial infiltrates. She, therefore, remained on mandatory volume controlled ventilation, heavy sedation and a continuous infusion of atracurium for the first ten days. The patient received specific COVID-19 treatment with remdesivir and dexamethasone. During her first 2 weeks in ITU, she was proned seven times in total with initially significant, but gradually decreasing clinical responsiveness to this intervention. The patient was deemed not to be a candidate for extracorporeal membrane oxygenation on the basis of premorbid function.
and a degree of improvement in gas exchange with proning. The patient’s secretion load substantially increased from day 9 when she developed a ventilator-associated pneumonia, and remained moderate even after this superimposed infection resolved by day 14.

During her third week of U admission, the patient showed an improvement in her gas exchange while supine, not needing any paralytic agents. As sedation was weaned, the patient was successfully switched to a spontaneous breathing pressure support mode with a positive end expiratory pressure (PEEP) of 8 cm H2O. On day 20, however, the patient suddenly became tachypneic, hypoxaemic, tachycardic and hypotensive. Ventilator waveforms revealed severe expiratory airflow obstruction, significant air trapping and ineffective inspiratory efforts.

INVESTIGATIONS AND DIFFERENTIAL DIAGNOSIS

Auscultation revealed air entry bilaterally and no wheezing. Arterial blood gas analysis showed the CO2 to be rising with stable oxygenation as reflected in the ABG in figure 1, which showed unchanged PaO2. An in-line suction catheter was passed with little difficulty through the ETT and drew back minimal aspirates of sputum. Despite this somewhat reassuring finding, we maintained a suspicion of possible partial airway obstruction, i.e. mucus plugging, tube kinking or tube dislodgement. Chest X-ray revealed no interval change or tube dislodgement (figure 2) and chest ultrasound did not suggest pneumothorax or lobar collapse. Afterwards, the patient was resedated, paralysed and ventilated with a volume controlled ventilation mode with the following settings: tidal volume (Vt) 420 mL, respiratory rate (RR) 18 breaths per minute, PEEP of 8 cm H2O, fraction of inspired oxygen 1.0, flow 60L/min.

Ventilator pressure waveforms showed mildly increasing peak inspiratory pressures (Ppeak) 45 cm H2O and a plateau pressure (Pplat) 30 cm H2O giving a total resistance (Rtot) 15 cm H2O/L/s. The end tidal CO2 trace and the expiratory flow trace on the ventilator screen persistently showed signs of severe expiratory airflow limitation.

The patient’s intrinsic PEEP (iPEEP) was 14 cm H2O. As we further reduced PEEP from 8 to 5 cm H2O in order to facilitate expiration, the patient showed worsening signs of air trapping, and an increase in iPEEP to 16 cmH2O. Subsequent her blood pressure began to drop. The signs of air trapping and expiratory airflow obstruction remained evident. A diagnosis of bronchospasm seemed unlikely because this patient had equal air entry bilaterally and only a mildly increased Rtot of 15 cm H2O. Therefore, another cause of deterioration was sought.

Taking all of this into account, the patient’s RR was reduced from 18 to 12 breaths per minute and her inspiratory to expiratory ratio was reduced from 1:2 to 1:4. This was done to facilitate expiration, CO2 clearance and to reduce dynamic hyperinflation with intrathoracic pressures suspected of potentially causing the haemodynamic compromise. However, none of these interventions managed to significantly improve the patient’s condition. Consequently, the decision was made to perform a bronchoscopy to visualise the airway from within.

Bronchoscopy revealed that a layer of pink mucus, which appeared to most of the team as mucosal tissue, had formed along the whole length of the tube. However, the blue line on the lower surface confirmed the scope was still inside the ETT (figures 3 and 4). Despite the confusing lining of the tube (figures 3–5). The smaller immobile first flap and the second free-moving flap were not opposed directly across the diameter of the tube but rather the larger flap was positioned slightly below the smaller one along the length of the tube (figure 4)—similar to the two leaflets of a foramen ovale in an atrial septal defect. Thus, by this mechanism we hypothesise the suction catheters and bronchoscope were able to pass through both mucinous flaps and retract back without disrupting the valvular formations and their obstructive airway effect.

TREATMENT

The ETT was changed under video laryngoscopy and the patient’s respiratory mechanics improved remarkably with no further signs of increased resistance inside the breathing circuit nor any expiratory airflow obstruction/airtrapping. Ventilator settings were adjusted to Vt 420 mL, RR 16, PEEP 8 cm H2O, flow 60L/min, generating Ppeak of 33 cm H2O and Pplat of 24 cm H2O, Rtot 9 cm H2O/L/s. The patient’s iPEEP was the...
COVID-19 is a serious, potentially fatal but avoidable complication that can be treated when recognised promptly. In addition, we would like to further analyse the reasons why mucinous flaps form in the first place and describe the effect they have on respiratory mechanics.

This patient had a high likelihood of developing mucus plugging for several reasons. First, long-term smoking is known to increase inflammation in the airway endothelium causing production of excess mucus. Second, patients with COVID-19 pneumonia tend to develop thick and sticky secretions. They often require thorough pulmonary hygiene including frequent suctioning, mucolytics and bronchodilators. Third, she had recently recovered from a ventilator associated pneumonia and continued to have ongoing moderate secretions, needing regular suctioning and circuit humidification. In our case these fairly nursing-intensive interventions could not always be guaranteed due to a combination of higher demands on staffing levels and an inconsistent skill-mix during the pandemic causing lower than usual nurse-to-patient ratios. However, the most important precipitating factor leading to this airway emergency was a lack of circuit humidification. Two days prior to this particular patient’s airway emergency, she was moved to a different part of the ITU in order to receive dialysis, and her ventilator was changed to an advanced ventilator model that did not have a built-in active humidification system. In lieu of this, a heat and moisture exchange (HME) was attached close to the patient end of the airway circuit. Even though the HME filters were changed daily, we believe this likely caused insufficient humidification contributing to the formation of thick and copious mucus deposits. HME filters are contraindicated in difficult-to-wean patients who have increased amounts of thick secretions and have higher minute volumes. After this emergency was resolved, the patient was switched back to the original ventilator which had an active pass-over humidification system applied to the circuit and she subsequently didn’t need any further tube changes.

Though humidification is a relatively simple component it had slipped into the cognitive blind spot of the ITU team. While HME filters are a simple and cost effective intervention that is appropriate for the majority of patients needing intermittent mechanical ventilation, there exists a patient population that need more effective humidification measures. Torrego et al describe in depth white and gelatinous secretions observed during bronchoscopy in intubated COVID-19 patients which often results in mucohematic plugs. They go on to suggest that the high incidence of this complication could be explained by the use of closed-circuit suction systems together with the HME instead of heated humidifiers.

With respect to respiratory mechanics, during bronchoscopy, we observed one of the two flaps was static throughout the respiratory cycle serving only to narrow the lumen by a small margin. The second flap, and greater in size, extended out from the inner wall of the tube and was fully mobile throughout the respiratory cycle serving only to narrow the lumen by a small margin. With positive pressure ventilation the flap would mobilise downwards towards the distal end of the tube flattening itself against the wall of the tube contributing minimally to the narrowing of the lumen. This explains why there was only a mildly increased resistive component during inspiration. However, in expiration the flap would swing upwards and extend out into the centre of the tube’s lumen creating a near complete seal over the diameter of the tube. With this mechanism that we observed how the mucinous flaps created a near perfect one-way valve resulting in severe expiratory airflow limitation with dynamic hyperinflation and increased iPEEP.

**DISCUSSION**

This case report highlights several very important learning points about the significance of secretion load in COVID-19, the importance of sufficient circuit humidification and a review of the respiratory mechanics seen with dynamic mucinous flaps inside an ETT.

COVID-19 can be a suppurative pathology which tends to form viscous and hard to suction gelatinous secretions and often result in muchohematic plugs. A case series recently published by Wiles et al described their experience with ETT obstruction among patients with ARDS due to COVID-19. They reported 11 intubated COVID-19 patients experiencing this complication and an overall incidence of 5.6%. With our clinical case, we wanted to demonstrate that ETT obstruction in patients with
Similar respiratory mechanics have been observed in other cases of airway obstruction in patients with COVID-19. It is well known that in spontaneously breathing patients with airflow obstruction, the application of PEEP or increasing the PEEP leads to improvements in work of breathing and reduces dynamic hyperinflation. In patients who are on controlled ventilation, the evidence suggests that application of low levels of PEEP compared with no PEEP may facilitate expiration by decreasing expiratory resistance and facilitating alveolar emptying. Interestingly, reducing the PEEP in our patient, who was paralysed and ventilated, had a negative effect. The subsequent bronchoscopy findings were key to deriving an explanation for this observation. We hypothesise that the lower PEEP most likely caused the larger second mucinous flap to swing towards the centre of the tube’s lumen more prominently during expiration; thus resulting in the more severe expiratory airflow limitation observed in our case. Therefore, in the context of dynamic airflow obstruction during non-spontaneous ventilation, where reducing PEEP precipitates worsening of the airflow obstruction, a potential diagnosis may well be mucinous flaps causing a dynamic effect.

**Learning points**

- Think about secretion build up in endotracheal tube of intubated patients with obstructed airflow with supplicative pathology.
- Easy passage of a suction catheter does not rule out partial or dynamic airway obstruction.
- Crew resource management is important in organising labour intensive (although simple) therapies such as nebulisers and mucolytics.
- Be mindful of scenarios where heat and moisture exchange is insufficient for the patient’s needs.
- In the context of airway obstructions during controlled mechanical ventilation, a reduction in positive end expiratory pressure might worsen dynamic hyperinflation and air trapping.

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Case reports provide a valuable learning resource for the scientific community and can indicate areas of interest for future research. They should not be used in isolation to guide treatment choices or public health policy.

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**REFERENCES**

1. Hazard D, Kainer K, von Cube M, et al. Joint analysis of duration of ventilation, length of intensive care, and mortality of COVID-19 patients: a multistate approach. BMC Med Res Methodol 2020;20:206.
2. King CS, Sahniani D, Brown AI, et al. Outcomes of mechanically ventilated patients with COVID-19 associated respiratory failure. PLoS One 2020;15:e0242651.
3. Khan MA, Khan ZA, Charles M, et al. Cytokine storm and mucus hypersecretion in COVID-19: review of mechanisms. J Inflamm Res 2021;14:175–89.
4. Yin W, Cao W, Zhou G, et al. Analysis of pathological changes in the epithelium in COVID-19 patient airways. ERJ Open Res 2021;7. doi:10.1183/23120541.00690-2020. [Epub ahead of print: 06 04 2021].
5. Fox SE, Akmatbekov A, Harbert JJ, et al. Pulmonary and cardiac pathology in African American patients with COVID-19: an autopsy series from new Orleans. Lancet Respir Med 2020;8:681–6.
6. Tang D, Comish P, Kang R. The hallmarks of COVID-19 disease. PLoS Pathog 2020;16:e1008536.
7. Amal J-M, Gamero A, Saoli M, et al. Parameters for simulation of adult subjects during mechanical ventilation. Respir Care 2018;63:158–68.
8. Tobin MJ, Jabrun A. Chapter 31. Mechanical Ventilation in Chronic Obstructive Pulmonary Disease. In: Tobin MJ, ed. Principles and practice of mechanical ventilation, 3 edn. McGraw Hill, 2013.
9. Torrego A, Pajares V, Fernández-Arias C, et al. Bronchoscopy in patients with COVID-19 with invasive mechanical ventilation: a single-center experience. Am J Respir Crit Care Med 2020;202:284–7.
10. Wiles S, Mireles-Cabodevilla E, Neuhof S, et al. Endotracheal tube obstruction among patients mechanically ventilated for ARDS due to COVID-19: a case series. J Intensive Care 2021;36:604–11.
11. Waldmann C, Rhodes A, Soni N. Oxford desk reference critical care chapter 1 respiratory therapy techniques, 2019.
12. Smith TC, Mainini UF. Impact of PEEP on lung mechanics and work of breathing in severe airflow obstruction. J Appl Physiol 1988;65:1488–99.
13. Kondili E, Alexopoulou C, Prininakis G, et al. Pattern of lung emptying and expiratory resistance in mechanically ventilated patients with chronic obstructive pulmonary disease. Intensive Care Med 2004;30:1311–8.
14. Canamé MF, Borge JB, Lucio MR, et al. Paradoxical responses to positive end-expiratory pressure in patients with airway obstruction during controlled ventilation. Crit Care Med 2005;33:1519–28.