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CASE REPORT

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Shock Due to an Obstructed Endotracheal Tube

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Abstract

Endotracheal tube obstruction by a mucus plug causing a ball-valve effect is a rare but significant complication. The inability to pass a suction catheter through the endotracheal tube with high peak and plateau pressure differences are classical features of an endotracheal tube obstruction. A case is described of endotracheal tube obstruction from a mucus plug that compounded severe respiratory acidosis and hypotension in a patient who simultaneously had abdominal compartment syndrome. The mucus plug was not identified until a bronchoscopic assessment of the airway was performed. Due to the absence of classical signs, the delayed identification of the obstructing mucus plug exacerbated diagnostic confusion. It resulted in various treatments being trialed whilst the patient continued to deteriorate from the evasive offending culprit. We suggest that earlier and more routine use of bronchoscopy should be employed in an intensive care unit, especially as a definitive way to rule out endotracheal obstruction.

Keywords: obstruction, endotracheal tube, mucus plug, bronchoscopy, ball-valve

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Case Presentation

A 63-year-old male, 170cm tall, weighing 69kg, was admitted to the Singapore General Hospital, Singapore, presenting with epigastric bloating and discomfort. He had a history of extensive smoking, hypertension, chronic kidney disease and Child C liver disease complicated by portal hypertension, ascites, oesophageal varies and gastropathy.

He was initially treated with antibiotics and ascitic drainage for culture-negative neutrocystic ascites. Unfortunately, he developed acute severe abdominal pain twenty-six days post-admission. A computed-tomography abdominal and pelvis investigation revealed a perforated duodenal ulcer, and subsequently, he went for an exploratory laparotomy with a roux loop mucosal patch, his first operation. Post-operatively, he was transferred to the surgical intensive care unit (ICU) for ventilatory and haemodynamic support.

On post-admission day twenty-eight, post-operative day two, it was noted that the patient had a high intra-abdominal pressure of 26mmHg, complicated by oliguric acute kidney impairment from abdominal compartment syndrome. This was managed medically with paralysis and renal replacement therapy. A duodeno-jejunal anastomotic leak was diagnosed on post-admission day thirty-five, post-operative day nine. He underwent a second exploratory laparotomy with tube diversion and primary repair of the wound dehiscence pedicled omentoplasty.

After significant improvement, he had definitive surgery consisting of primary exclusion and roux loop gastrojejunostomy, his third operation, five days after the second operation, on post-admission day forty. Intra-operatively there was significant blood loss of 5.5L, and he received seven units of packed red cells, two units of pooled platelets and six units of fresh frozen plasma. Ventilatory support was uneventful intra-operatively, with volume-controlled ventilation generating 400mL of tidal volume with peak airway pressure of 22cm-H2O. Noradrenaline (Labaratoire Aguettant, Lyon, France) infusion up to 0.4mcg/kg/min intraoperatively was initiated. At the end of the operation, he was returned to the ICU where a noradrenaline (Labaratoire Aguettant, Lyon, France) infusion of 0.03 mcg/kg/min was continued.

Two hours after returning from his third operation, ventilation was difficult. His peak pressure (Ppeak) started to rise, up to 50cmH2O, whilst only producing less than 200mL of tidal volume on volume-controlled ventilation and his end-tidal carbon dioxide rose to 87mmHg.

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To improve the patient's ventilator dyssynchrony, he was paralysed with an atracurium (Zyfas Medical Co, Singapore) infusion running at 0.3-0.6ml/kg/hr, titrated to a train-of-four count of 2. In addition, he was sedated with propofol (Fresenius Kabi, Graz, Austria) 2-4mg/kg/hr, titrated to a bispectral index of 40-60.

The ventilator pressure-time curve demonstrated increased peak and plateau pressures, above 35cmH_2O, without any rise in the difference between the peak and plateau pressures.

On examination, his chest was clear bilaterally, with generalised poor air entry throughout. Multiple endotracheal tube suctioning yielded minimal secretions without meeting any resistance during the passage of the catheter. An arterial blood gas was done and revealed pH 6.99, pCO2 106mmHg, pO2 125mmHg and base excess -6. A chest X-ray was performed but did not show any apparent cause for this acute deterioration. The intra-abdominal pressure at the time was 31mmHg. Noradrenaline (Labaratoire Aguettant, Lyon, France) was increased to 0.3mcg/kg/min, and vasopressin (Fresenius Kabi, Canada) was started at 1.8 units/hr to maintain adequate mean arterial pressure (MAP) targets.

His haemoglobin seemed to be dropping since his operation: 8.9g/dL immediately post-operatively to 7.7g/dL when he was deteriorating. This raised the suspicion of an intra-abdominal haemorrhage resulting in abdominal compartment syndrome and ventilatory difficulties.

He underwent another exploratory laparotomy, his fourth, on post-admission day forty-one, a few hours after the third operation; only a small amount of intra-abdominal bleeding with blood clots and moderate generalised bowel oedema was found.

Opening of the peritoneum showed some improvement in ventilation and haemodynamics. Ppeak was 45cmH2O delivering 290mL tidal volume at the start of the operation and improved to 38cmH2O with a tidal volume of 350mL after opening the peritoneum. Vasopressin (Fresenius Kabi, Canada) was stopped, and noradrenaline (Labaratoire Aguettant, Lyon, France) was weaned to 0.1mcg/kg/min by the end of the operation to maintain a MAP above 65mmHg.

Post-operatively, although improved, the Ppeak and respiratory acidosis persisted despite an improvement in intra-abdominal pressure to 22mmHg. Ppeak remained high at 35cmH2O, and the arterial blood gas blood test showed a pH 7.164, pCO2 72.8mmHg, pO2 152mmHg and BE -4.

A bedside bronchoscopy was performed for visual assessment of the endotracheal tube and respiratory system. A large mucus plug was found at the tip of the endotracheal tube that oscillated with each delivered ventilator breath. No other airway abnormalities were found. Retrospectively, on review of the pre-operative ventilator flow-time curve, as shown in Fig 1, the curve was seen to depict a slow, constant expiratory flow rate that did not reach zero at the end of expiration; this was suggestive of the presence of an expiratory flow limitation resulting from a ball-valve effect from the mucus plug.

The endotracheal tube was exchanged on post-admission day forty-one, immediately after the fourth operation. It resulted in an immediate improvement in the Ppeak from 35cmH2O with a tidal volume of 350mL to 18cmH2O with a tidal volume of 450mL.

The ventilator flow-time curve returned to normal (Fig 2.), and the noradrenaline (Labaratoire Aguettant, Lyon, France) was stopped within minutes.

Arterial blood gases blood test was repeated one hour later, which showed pH 7.42, pCO2 39.1mmHg, pO2 76.1mmHg and BE 0.5.

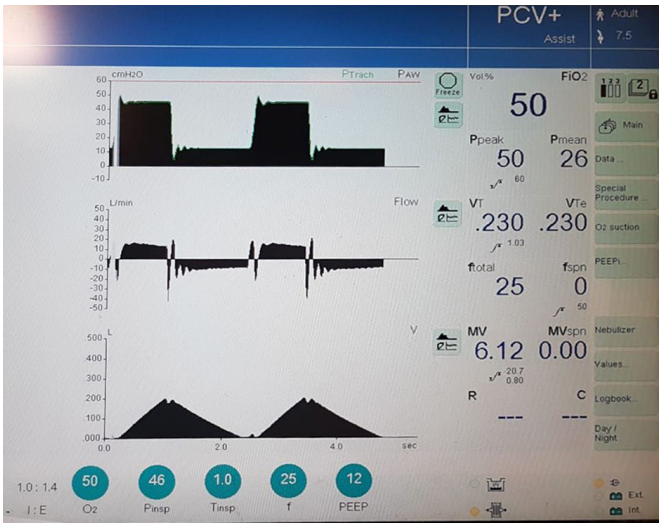

Fig. 1. Image of ventilator dashboard during clinical deterioration just before the emergency temporary abdominal closure. Ventilation with pressure-controlled ventilation with a set inspiratory pressure of 46mmHg, inspiratory time of 1 second and positive end-expiratory pressure (PEEP) of 12mmHg. The tidal volume delivered was 230mL. The flow-time curve revealed a waveform suggestive of an expiratory flow limitation.

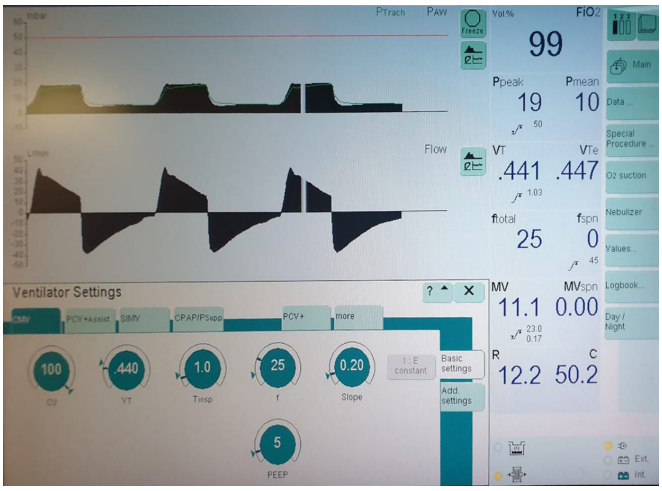

Fig. 2. Image of ventilator dashboard immediately after the exchange of endotracheal tube (ETT). Ventilation with volume-controlled ventilation with a set tidal volume of 440mL, inspiratory time of 1 second and positive end-expiratory pressure (PEEP) of 5mmHg. The peak airway pressure (Ppeak) was 19cmH2O. Thus, the flow-time curve waveform is consistent with a normal expiratory phase.

Unfortunately, over the next ten days, the patient did not improve, and his state was further complicated by gastrointestinal bleeding from the breakdown of the surgical anastomosis. He developed overwhelming sepsis with multiorgan failure and subsequently died fifty days after hospital admission.

Discussion

This case presents a complex clinical scenario of inadequate mechanical ventilation from two different pathologies: abdominal compartment syndrome and mucous plugging of the endotracheal tube with ball-valve effect. Ventilatory difficulties with high Ppeak is commonly caused by poor lung compliance or increased airway resistance. Difficult ventilation in the patient was due to reduced chest wall compliance, caused by abdominal compartment syndrome secondary to bowel oedema, and poor lung compliance caused by air trapping secondary to the mucus plug. While the temporary closure of the abdomen helped improve Ppeak, it did not completely resolve the problem. The ball-valve effect caused by the thick mucus plug at the tip of the endotracheal tube resulted in hyperexpanded lungs with raised intrathoracic pressures. It was likely the main cause of the ventilatory and circulatory problems. This is supported by the immediate improvement in Ppeak and blood pressure once the endotracheal tube was exchanged.

Mucus plug formation is not uncommon in tracheotomised ICU patients. They result from abnormal secretions of salt and water, increased mucin production, and mucus infiltration with inflammatory cells in combination with decreased mucus clearance [1]. Invasive ventilation increases the risk for sputum retention as mucociliary clearance is impaired. Furthermore, dry gases cause the mucosa to produce more mucus and increase its viscosity, forming a mucus plug within the endotracheal tube.[1,2] Humidification during mechanical ventilation helps prevent the drying out of secretions and, hence, thick mucus plugs [3]. Routine endotracheal tube suctioning can help remove excess mucus, whilst kinetic therapy and chest physiotherapy can help with mucus clearance [2].

The ball-valve effect can cause many downstream effects, as evident in the present case but reaching a diagnosis is not straightforward.

Investigations should be centred around looking for signs of hyperinflation. Clinically, diminished or absent breath sounds may be heard, along with hyper-resonance and reduced chest excursion. On a chest X-ray, hyperinflation of the lungs can be seen as hyperlucency of lung fields, reduced pulmonary markings, flattening of the diaphragm, rib flaring, and a mediastinal shift depending on the location of the obstruction.

However, it has been reported that 30-40% with a ball-valve type of obstruction will have a normal chest X-ray [4,5].

In the present case, the chest X-ray did not show any hyperinflation or flattening of the diaphragm, possibly due to the co-existing abdominal compartment syndrome. An ultrasound could also be used to look at diaphragm kinetics using M-mode to assess the ratios of diaphragmatic excursion to the volume of respiration [6].

Interpretation of the ventilator pressure and volume curves assists in the diagnosis of ventilation problems. Typically, there is a marked difference between Ppeak and plateau airway pressures in airway obstruction, despite normal plateau pressures seen on pressure-time curves. However, this was not present in the present case due to the nature of the mucus plug. However, the flow-time curve (Fig.1) did reveal a waveform suggestive of an expiratory flow limitation. This atypical presentation of ventilator flow-time curves of an endotracheal tube obstruction could be explained by the auto peak end-expiratory pressure effect created by the mucus plug.

Conventionally, the ability to pass a suction catheter down the endotracheal tube would rule out an endotracheal tube obstruction. However, in this case, it failed to identify the obstruction, which contributed to a delay in diagnosis that was only confirmed by bronchoscopy. The usual indications for fibre optic bronchoscopy in an ICU include broncho-alveolar lavage for culture collection in pneumonia and treatment in patients who have aspirated; foreign body retrieval; haemostasis during haemoptysis; evaluation of airway injury and tracheal visualisation during percutaneous tracheostomy [7]. With the increasing availability and clinical expertise with bronchoscopy, intensive care physicians should be encouraged to perform bronchoscopies more routinely.

Conclusion

Mucus plugging is not an unusual occurrence in an ICU. The current case indicates that significant clinical implications can occur with an undiagnosed ball-valve effect. The ability to pass a suction catheter down the ETT did not rule out airway obstruction, and subtle, atypical obstructive patterns of ventilator curves can easily be missed.

The cause of the patient's clinical deterioration was only identified following bronchoscopy.

Therefore, it is proposed that in the event of unexplained high airway pressures, it would be advisable to examine the endotracheal tube and airway with a fibre optic bronchoscope at an early stage.

Conflict of Interest

None to declare.

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