Case report

Airway pressure release ventilation for treatment of life-threatening adult respiratory distress syndrome secondary to fat embolism syndrome

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Abstract

Airway pressure release ventilation is a novel mode of positive pressure ventilation that has advantages over low tidal volume, assist control ventilation in patients with acute respiratory distress syndrome (ARDS). It also helps to meet the goals of ventilatory management of patients with ARDS such as lung protection strategy. We report a patient with trauma who developed fat embolism syndrome (FES) followed by severe life-threatening ARDS and successfully managed with airway pressure release ventilation.

Key words: Airway pressure release ventilation, Acute respiratory distress syndrome, Fat embolism syndrome.

Introduction

Airway pressure release ventilation (APRV) is a relatively newer mode of positive pressure ventilation that became commercially available in US in mid 1990s but is gaining more attention in the recent years. In conventional modes of ventilation the respiratory cycle begins at a baseline pressure and is raised periodically to provide the pressure gradient for ventilation. In APRV, the patient is maintained at an elevated airway pressure and is released from time to time to achieve tidal ventilation *(Figure 1)*.

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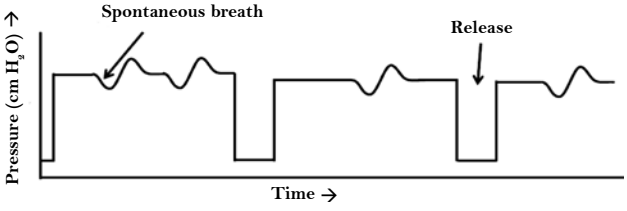

Figure 1: Pressure-time scalar of airway pressure release ventilation.

The elevated baseline pressure increases mean airway pressure thus facilitating oxygenation. The timed release produces a pressure gradient along which gases flow outwards aiding carbon dioxide clearance. APRV first described by Stock and Downs in 1987, is a time-triggered, pressure limited, time- cycled mode of ventilation that allows unrestricted spontaneous breathing throughout the entire ventilatory cycle.[1] APRV is a recognised mode of ventilation in trauma patients with ARDS.[2] APRV helps to meet the goals of ARDS management wherein alveolar recruitment is maximised due to increased mean airway pressure. At the same time, transalveolar pressure gradient is limited and the risk of barotrauma is decreased.[3]

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Thus, APRV can be described as a lung protection strategy that can minimise lung injury seen with mechanical ventilation.[4] We report a case of a patient with alleged history of road traffic accident and had open fracture of shaft of right tibia, fibula, closed fracture of right femur who developed fat embolism syndrome (FES) and severe life-threatening ARDS who was successfully managed with APRV.

Case report

A 22 year old man presented to trauma triage 9 hours following motor cycle accident. He was fully conscious and oriented. He was in severe agony, tachycardic (140/min) but with a stable blood pressure (130/90 mm Hg), respiratory rate (22/min) and oxygen saturation (98%). He was diagnosed to have open fracture of shaft of tibia and fibula and closed fracture of right femur that was confirmed by X-ray. Toe movements, sensation and circulation were intact. Examinations of all other systems were unremarkable. Intravenous lines were established. Crystalloids and analgesics were given. Routine investigations were done.

Three hours later, the patient was shifted to orthopaedic high dependency unit (HDU) for further manage-ment and monitoring where his oxygen saturation was found to be decreasing needing oxygen therapy. Fat embolism was suspected and an early fixation of the fracture was planned. He was shifted to the operation theatre (22 hours after admission) for external fixator application under general anaesthesia. The procedure was uneventful with stable haemodynamics but the oxygen saturation was low. In view of altered sensorium and hypoxia in arterial blood gas analysis (ABG), the patient was shifted to Intensive Care Unit (ICU), suspecting a fat embolism syndrome (FES).

Initially he was ventilated with volume control ventilation. As his peak pressures were increasing the mode was switched over to pressure controlled ventilation with volume guarantee (PCV-VG) with fractional inspired oxygen concentration (FiO_2) 0.8, positive end expiratory pressure (PEEP) 10 cm H_2O, Pressure limit (Pmax) 35 cm H_2O with target tidal volume (V_T) of 500 ml. With this ABG reports showed normal acid base status and acceptable oxygenation [PaO_2/FiO_2 (P/F) ratio = 253].

Next day, the patient was shifted to operation theatre using a transport ventilator for interlocking and nailing of right fibula and right tibia during which he desaturated to 58% even on FiO_2 1.0 and PEEP 15 cm H_2O. He was tachycardic and hypertensive. He was suspected to have a fresh episode of fat embolism and since he was so critical, was shifted back to ICU postponing the surgery to another day. Ventilation was resumed in PCV-VG mode with FiO_2 1.0, PEEP 20 cm H_2O, V_T 400 ml, respiratory rate 20/min, Pmax 40 cm H_2O. ABG done on these settings showed uncompensated respiratory acidosis with critical oxygenation (P/F) ratio = 100. Even after ventilating with baby lung concept and open lung concept for approximately six hours, oxygenation did not improve and $PaCO_2$ was 100 mm Hg. At this point of time, patient was switched over to APRV mode of ventilation (Dräger Evita™ 2 Dura: Dräger Medical Inc., Lübeck, Germany) with a P_{high} 35 cm H_2O, P_{low} 15 cm H_2O, Time high : Time low (T_H : T_L) :: 2:1, release rate 20/min and FiO_2 of 1. An hour after initiation of APRV, ABG showed normal acid base status with poor oxygenation (pH = 7.44, $PaCO_2$ = 44 mm Hg, PaO_2 = 64 mm Hg, HCO_3^- = 25 mmol/L). Subsequent ABG values showed improved oxygenation and ventilation with APRV mode *(Figure 2)*. This was also reflected in chest X-ray *(Figure 3)*.

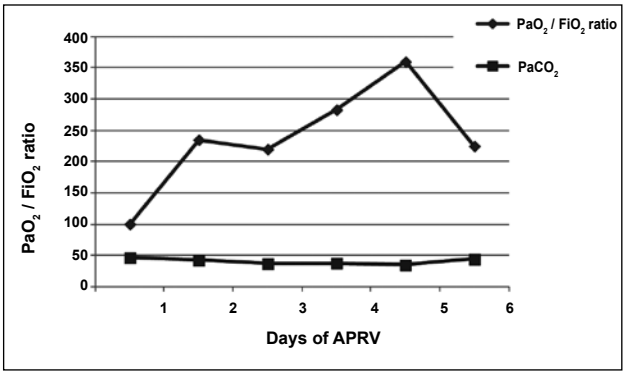

Figure 2: Changes in PaO_2/FiO_2 ratio and arterial carbon dioxide tension after initiation of APRV.

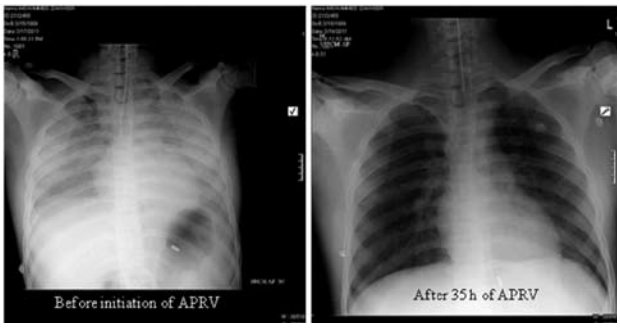

Figure 3: Chest X-ray before initiation of APRV (left) and 35 hours after initiation of APRV (right).

The patient was sedated and paralysed for 4 days. Sedation was continued with morphine and by the seventh day, the patient was weaned to biphasic positive airway pressure (BIPAP) mode. Gradually weaning over four days to P_{high} of 24 cm H_2O and P_{low} of 10 cm H_2O and $T_{high} : T_{low}$ :: 1:1.5 was done. He kept improving steadily and interlocking nailing of right tibia and fibula could be done on the eleventh day after commencement of APRV. Following the procedure, he was again received in ICU and was on BIPAP mode with P_H of 10 cm H_2O and P_L of 6 cm H_2O and $T_H : T_L$ :: 1:2. He could be rapidly weaned to pressure support ventilation (PSV) with FiO_2 0.5, PEEP/PSV of 6/10 cm H_2O within four hours of surgery. He was monitored for another hour and was extubated. He was put on 60% oxygen by Venturi mask. Arterial blood gas done on same FiO_2 next day showed normal acid base status with deterioration in oxygenation (P/F ratio 144). This could be managed with continued oxygen therapy and deep breathing exercises. He could be weaned to 4 L/min of oxygen through nasal prongs and was shifted to orthopaedic HDU for further management.

Discussion

Although APRV was described way back in 1987, it has received increased attention in the recent years as a mode not only providing lung protection strategy but also has additional advantage of allowing spontaneous breathing during positive pressure ventilation. This patient had to be sedated and paralysed initially and probably improved only due to the increased mean airway pressure and the pattern of ventilation. The patient was very critical and at risk of death. The options for rescue manoeuvres would have been prone ventilation or high frequency oscillation in our setup. Nitric oxide therapy and extracorporeal membrane oxygenation are not available at our hospital. We preferred to attempt APRV before attempting prone ventilation or HFOV for ease of management.

APRV has been reported to provide better oxygenation with lower peak pressure,[3,5,6] better haemodynamics and fewer ICU days.[7] In this report, we have illustrated yet another case of fat embolism and severe ARDS rescued with the use of APRV.

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