

Severe Pediatric Acute Respiratory Distress Syndrome Due to Scrub Typhus: Successful Ventilation with Airway Pressure Release Ventilation Mode after Becoming Refractory to Protective Ventilation

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Abstract

Scrub typhus can affect lungs from mild illness like pneumonitis to a severe illness like acute respiratory distress syndrome (ARDS). Such patients may be very challenging to treat when their hypoxemia becomes severe and refractory to treatment. Main treatment is supportive in terms of mechanical ventilation. In adult ARDS, low tidal volume (TV) ventilation has been recommended, but there is no consensus on most effective ventilation mode in children. We present a case of a 12-year-old girl who developed severe ARDS (PO_2/FiO_2 ratio - 58), refractory to low TV ventilation. There was a rapid improvement in oxygenation on the application of airway pressure release ventilation (APRV) mode within ½ h. She was successfully ventilated and weaned off the ventilator over 5 days. This case highlights the utility of APRV mode of ventilation as a rescue therapy for severe refractory ARDS in children.

Keywords: Acute respiratory distress syndrome, airway pressure release ventilation, pediatric, scrub typhus

INTRODUCTION

Acute respiratory distress syndrome (ARDS) is one of the serious complications of scrub typhus infection. It can result in severe hypoxemia refractory to mechanical ventilation. There are few options for mechanical ventilation for such situations such as high frequency oscillation ventilation and extracorporeal membrane oxygenation. The role of airway pressure release ventilation (APRV) mode is not clear, especially in children. In this case report, we describe a young girl who was successfully treated with APRV mode of ventilation after the failure of protective ventilation strategy.

CASE REPORT

A 12-year-old girl was admitted to our Pediatric Intensive Care Unit due to respiratory distress from pneumonia. In addition, she also had hepatitis and thrombocytopenia. An eschar was seen on her back. Intravenous azithromycin was started on clinical suspicion of scrub typhus. On the 3rd day, she deteriorated and developed hypoxemia. Her SpO_2 was

86% on an oxygen flow of 8 l/min by simple mask. She was started on continuous positive airway pressure (positive end expiratory pressure (PEEP) up to 10 cmH₂O) but didn't show any improvement. Now, her PO_2/FiO_2 (PF) ratio was 58, more than two quadrants were affected on chest X-ray, had received a PEEP of 10 cmH₂O and echocardiography was normal [Figure 1]. A diagnosis of severe ARDS was made. She also developed shock. She was ventilated with FiO_2 -100%, positive inspiratory pressure (PIP) - 22 mbar, PEEP - 5 mbar, rate - 18/min, and Ti - 1 s for persistent hypoxemia and received dopamine with noradrenaline for shock. We had to sedate her for patient-ventilator asynchrony. We had to increase gradually PIP and PEEP to 34 mbar and 17 mbar,

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respectively, but PO_2 did not improve. Now, the SpO_2 and PF ratio were 90% and 61, respectively. Ventilation monitoring showed a P_{peak} : 34–36 mbar; compliance: 14.5–17 ml/mbar; mean airway pressure (MAP): 19 mbar; $P_{plateau}$: 35 mbar, and tidal volume (TV): 149–176 ml (6–7 ml/kg). Reducing the TV further, resulted in lower SpO_2 and pH. Increasing PEEP led to worsening in shock. After 18 h, PO_2 dropped to 54 mmHg. We changed her ventilation mode to APRV. Lung injury score was 3.5 before starting APRV. Initial settings were P_{high} - 26 mbar, P_{low} - 0, T_{high} - 4 s, and T_{low} - 0.4 s. After ½ h, the SpO_2 increased from 90% to 94%; and after 1 h, it increased to 97% with PO_2 of 75.6 mmHg. The TV, compliance, and MAP increased to 10 ml/kg, 26 ml/mbar, and 23 mbar, respectively. P_{peak} decreased to 28 mbar [Figure 2]. We could reduce FiO_2 to 80%. Sedation was stopped as there was no asynchrony. Consistent improvement was observed, and after 3 days, weaning was started by reducing P_{high} by 2 and increasing T_{high} by 0.5 (drop and stretch technique). Shock also improved, and vasopressor medications were stopped. She showed mild-moderate intercostal retractions when reached at settings of P_{high} - 18 mbar and T_{high} - 6 s. We stopped the ventilator weaning at this stage and maintained the settings at a little higher levels (P_{high} - 20 and T_{high} - 6) for 12 h. Chest X-ray showed complete aeration [Figure 3]. Weaning was again tried, and this time, we could gradually reduce the settings to P_{high} - 6 and T_{high} - 10. She was put on T-piece which she tolerated well without any respiratory distress. After 2 h, she was extubated successfully, kept under observation for 24 h and shifted to the ward from where she was discharged after 2 days. Meanwhile, her serology showed elevated OX-K titers (>1:160) suggestive of scrub typhus. She was followed up for 4 months after discharge and showed good health.

DISCUSSION

Scrub typhus is a rickettsial infection caused by *Orientia tsutsugamushi* transmitted by larval trombiculid mites. In humans, it often causes multisystem disease. Pulmonary complications include pneumonitis, pleural effusion, and ARDS. ARDS as a complication of scrub typhus is rare. The scant pediatric literature shows that 1.1%–9.3% children with scrub typhus may develop ARDS.^[1,2] Adult literature shows that its incidence is more in patients who are diagnosed late.^[3] Organ damage in scrub typhus is due to microvascular endothelium damage resulting in vasculitis, perivasculitis, and increased microvascular permeability.^[4] The role of immune mechanism without vasculitis is also likely as shown in lung biopsy reported by Park *et al.*^[5] Our patient was affected severely and became refractory to low TV ventilation. It showed rapid and sustained improvement in oxygenation on the application of APRV mode of ventilation. APRV increases oxygenation by giving a continuous high pressure (P_{high}) to maintain MAP and thus resulting in alveolar recruitment.^[6] This pressure is released for very brief time to facilitate tidal ventilation (T_{low}). Asynchrony was less as the patient could breathe spontaneously throughout ventilator breath. This resulted in less or no sedation and

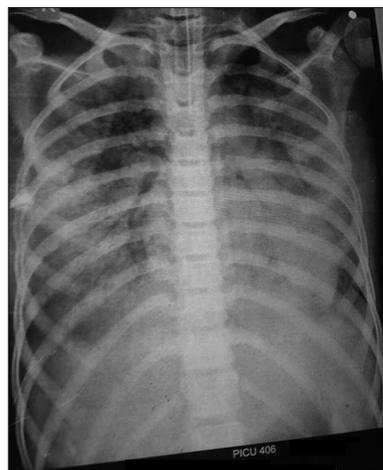


Figure 1: Chest X-ray showing more than two lung quadrants affected used for lung injury score

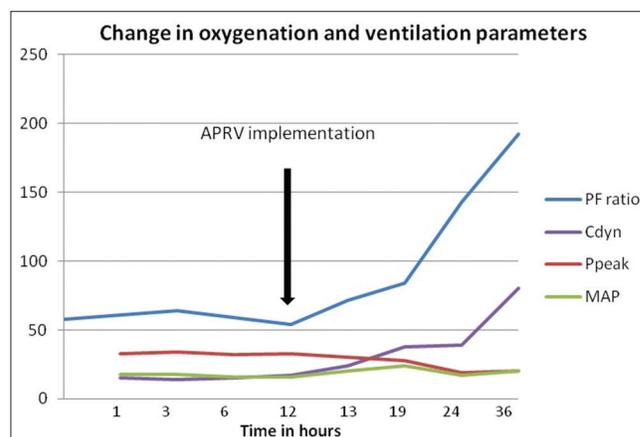


Figure 2: Graph showing better oxygenation and ventilation parameters after implementation of airway pressure release ventilation mode in same patient

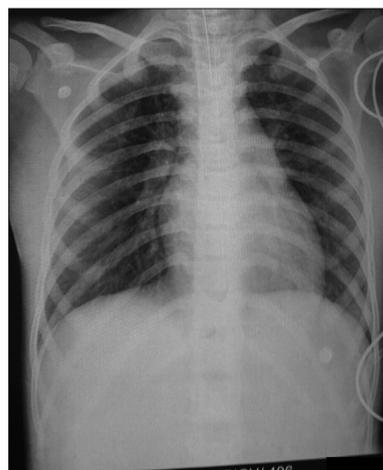


Figure 3: Chest X-ray showing complete aeration of the lungs after implementation of airway pressure release ventilation mode

muscle relaxant use. Moreover, patient self-breathing leads to preservation of respiratory muscles and ventilation of

dependent lung areas reducing V/Q mismatch. It limits peak pressure and increases compliance.^[7] We observed that shock state did not worsen with APRV rather it improved, and vasopressors could be tapered in 48 h. It has been postulated that abdominal pressure is increased, and pleural pressure is decreased during spontaneous breathing (when on APRV) which encourages venous return. Another very important observation from animal studies is that APRV may reduce the risk of ventilator-induced lung injury and so may be more protective than other modes of ventilation.^[8] In this case, APRV mode of ventilation was the game changer. APRV mode resulted in survival of the patient with a good mental and physical outcome. This case is unique for two reasons: one, scrub typhus is a rare cause of ARDS in children, and the other, there is a limited experience on the role of APRV mode in severe ARDS in children. To conclude, APRV mode is an effective ventilation strategy to improve oxygenation rapidly in pediatric patients with severe ARDS. It can be used as a rescue therapy in severe ARDS when it becomes refractory to conventional low tidal volume ventilation.

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Conflicts of interest

There are no conflicts of interest.

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