**Vecuronium in tuberculosis: A rare case report of reversible quadriparesis**

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**ABSTRACT**

Tuberculosis is a major health burden worldwide. The national treatment regimens for tuberculosis (TB) patients recommend the use of the five first lines anti TB drugs: isoniazid (INH), rifampicin (R), ethambutol (E), pyrazinamide (P) and streptomycin (S). Maintaining of oxygenation are very much challenging in tuberculosis patients associated with Acute Respiratory Distress Syndrome (ARDS). Often we need muscle relaxation with adequate sedation for maintaining oxygen saturation and lung recruitment. Skeletal muscle weakness has a confusing list of names and syndromes, including Acute Quadriplegic Myopathy Syndrome (AQMS), floppy man syndrome, critical illness polyneuropathy (CIP), and acute myopathy of intensive care. In disseminated tuberculosis with ARDS, we recommend the use of short-acting muscle relaxant drugs like cisatracurium whose metabolism not depends upon the liver. Interrupting the vecuronium infusion (vecuronium holiday) as its action was potentiated by streptomycin and corticosteroid which may result in the development of Critical Illness Polyneuro Myopathy (CIPM). Targeting Train of Four (TOF) of two rather than zero of four has been shown to be beneficial for a period of fewer than 48 hours.

**INTRODUCTION**

Tuberculosis is a major health burden worldwide. The national treatment regimens for TB patients recommend the use of the five first lines anti TB drugs Isoniazid (INH), rifampicin (R), ethambutol (E), pyrazinamide (P) and streptomycin (S). High hepatotoxic potential first-line antitubercular drugs are INH, R and P sometimes may need discontinuation. Tuberculosis is being increasingly recognized as a cause of acute respiratory distress syndrome. Maintaining oxygenation are very much challenging in a patient of tuberculosis associated with ARDS. Many times we need muscle relaxation with adequate sedation for maintaining oxygen saturation and lung recruitment. The selection of muscle relaxant in patients taking antitubercular treatment (ATT) with deranged liver function is challenging as it not only decreases metabolism of muscle relaxant but also potentiates its action due to the accumulation of metabolites. Skeletal muscle weakness has a confusing list of names and syndromes, including Acute Quadriplegic Myopathy Syndrome (AQMS), floppy man syndrome, Critical Illness Polyneuropathy (CIP) and acute myopathy of intensive care, etc.

**CASE PRESENTATION**

We report a diagnosed case of miliary tuberculosis (Figure 1) of a 16 years-old normotensive, non-diabetic female patient on HRZE regimen who developed severe ARDS. At the time of admission to Intensive Care Unit (ICU), she was conscious, oriented, febrile, cyanosed, tachypneic with a respiratory rate of 38/min, pulse rate of 104 beats/min, temperature of 101.8°F, blood pressure of 138/78 mmHg, and oxygen saturation of 78% on 15 litres per minute of oxygen by facemask. Systemic examination revealed bilateral coarse crepitations in the lungs. Rest of the examination was unremarkable.

Initial investigations revealed hemoglobin 9 g/dl and leucocyte count 23 x10^9/L (84% neutrophils and 20% lymphocytes), platelet count of 140,000/mm³, blood urea of 21.20 mg/dl, serum creatinine 0.29 mg/dl, potassium 4 mEq/L, sodium 133 mEq/L, calcium 7.9 mmol/L, serum bilirubin 0.30 mg/dl, total protein 5.53 g/dl, SGOT 281 U/L, SGPT 168 U/L, and ALP 222 U/L. ABG showed pH 7.62, pO₂ 34 mmHg, pCO₂ 36 mmHg, HCO₃ 20.8 mEq/L on non-rebreathing mask with O₂ at 15 L/min, depicting Type 1 respiratory failure. PF ratio (pO₂/FiO₂) was <50%. ECG showed sinus tachycardia. Chest x-ray (Figure 2) showed bilateral dense reticulonodular pattern interspersed with diffuse fine infiltrates.

The patient was managed with invasive mechanical ventilation and broad-spectrum antibiotics and steroids. Influenza retroviral and other virological serologies were negative. Blood and urine culture came sterile. There was no evidence of fungal infection. Bedside echo result was normal.

Tracheal aspiration showed strong positivity for Mycobacterium tuberculosis but Mantoux test came negative. Later on we found increased peak

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airway pressure (>38 cmH₂O), ventilator dysynchrony, and SpO₂ <80% so we sedated and paralyzed the patient as a lung protective strategy by using infusion midazolam (2 mg/hour) and vecuronium (0.08 mg/kg) bolus followed by infusion of 0.05 mg/hour by targeting a TOF zero of four until lung condition improved.

After 48 hours we discontinued both vecuronium and midazolam. We change the ventilator settings to weaning mode. Three to four hours following stoppage of infusion we performed manual muscle testing which showed shoulder and elbow strength (grade 2/5) and wrist extensor and flexors strength (grade 2/5). In the lower extremities, hip strength was grade 2/5, knee extensors grade 2/5, and ankles grade 2/5. No cranial nerve or sensory nerve involvement and fasciculation was noted with normal reflexes and muscle tone. Peripheral nerve weakness was the diagnosis. Serial muscle enzymes were followed and maximum creatinine kinase (CK) was 65 (normal range = 60-220). She was started on bedside physical therapy and occupational therapy and she regained muscle strength in all four limbs by the 15th day.

DISCUSSION

As per our knowledge, this is the first case reported in miliary tuberculosis patient on ATT developing ARDS on receiving vecuronium infusion developed quadriparesis. Careful considerations must be held before using muscle relaxant especially in ARDS patients with disseminated miliary tuberculosis receiving anti-tubercular drugs with deranged liver function, as it not only decreases metabolism and but the action of vecuronium was potentiated by streptomycin and corticosteroid which may result in the development of CIPM. Oxygenation of the lung can be improved by reducing oxygen consumption⁴ and by providing homogenous distribution of positive-end expiratory pressure (PEEP) and tidal volume which prevents barotrauma and worsening of ARDS.

Forel et al⁵ demonstrated neuromuscular blocking agents (NMBAs) may decrease the inflammatory response associated with ARDS. NMBAs may cause harmful effects, particularly Critical Illness Polyneuro Myopathy (CIPM) when used for prolonged periods or in septic shock. In our case we did not perform electromyography (EMG) and nerve conduction velocity (NCV) tests because of their unavailability at our institute, but neurological examination reveals a global motor deficit affecting muscles in both the upper and lower extremities and decreased motor reflexes with preserved sensory reflexes and extracranial muscle function, so we probably diagnose our case as CIPM.

Figure 1  Chest XRay showing miliary tuberculosis

Figure 2  Chest XRay showing ARDS Changes
Arroliga et al. international study of mechanically ventilated patients reported the median duration of use of two days. In acute lung injury and ARDS patients, however, NMBAs are used for even shorter periods. This study concluded that the duration of paralysis was the more important depth of paralysis. CIPM incidence is 25% but CIPM was associated with corticosteroid administration and the duration of mechanical ventilation. Danon et al. described that asthmatic patients treated with vecuronium and steroids that developed flaccid quadriplegia. Quadriplegia and marked atrophy were treated in a rehabilitation setting had an excellent outcome and the most likely explanation is steroid-induced acute myopathy, facilitated by neuromuscular blocking agents.

CONCLUSION

In disseminated tuberculosis with ARDS, we recommend the use of short-acting muscle relaxant drugs like cisatracurium whose metabolism does not depends upon the liver. Interrupting the vecuronium infusion (vecuronium holiday) is recommended as its action was potentiated by streptomycin and corticosteroid which may result in the development of CIPM. Targeting a TOF of two of four rather than zero of four has been shown to be beneficial and for a period of fewer than 48 hours.

REFERENCES