Short Communication

Extubation failure in PICU: The Preventive Strategies and Lessons Learnt

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ABSTRACT

Mechanical ventilation has always played a pivotal role in saving lives in pediatric critical care. But as indications for the same have been elaborated in literature, weaning is still a challenging task. A structured weaning protocol is required to prevent extubation failure in a critical care setting. Prolonged mechanical ventilation, extended sedative use, post-extubation stridor (PES), and diminished respiratory muscle strength are major causes of failed extubation. To tackle its impact, many new techniques, indices, and modalities are being developed. Using non-invasive ventilation for weaning, peri-extubation steroids, minimal sedation, and nutrition maintenance are some of the strategies that have been shown to improve the ventilation outcomes. Diaphragmatic ultrasonography has emerged as a new non-invasive technique to test diaphragm strength and can be used to assess readiness for successful extubation. Integrating knowledge about risk factors, applying defined tests and maneuvers, and using different parameters and ultrasonography to monitor diaphragm function can improve the chances of successful extubation.

Key words: Critical care, extubation failure, diaphragm ultrasound, pediatric intubation

Mechanical ventilation is a crucial intervention in pediatric critical care, pivotal in addressing respiratory illnesses, neurological dysfunction, and post-surgical recovery, thereby enhancing patient survival rates. However, both premature and delayed extubation pose risks and complications. Prolonged mechanical ventilation, while sometimes necessary, is linked to elevated mortality, heightened morbidity, increased infection rates, higher healthcare costs, and prolonged stays in the Pediatric Intensive Care Unit (PICU). Therefore, prioritizing safe and timely extubation is paramount, aiming to optimize patient outcomes and minimize adverse effects.

Extubation failure, a complex phenomenon, is influenced by diverse factors such as inadequate respiratory drive, compromised respiratory muscle function, and excessive inspiratory load. It is frequently associated with genetic syndromes, prolonged mechanical ventilation, extended sedative use, post-extubation stridor (PES), and diminished respiratory muscle strength. Despite meticulous care and interventions, regrettably, 15-20% of pediatric patients experience extubation failure [1, 2].

In this study, we discuss the spectrum of unusual and less discussed cases encountered during the extubation journey in the PICU.

CASE PRESENTATIONS

This study was done in the Department of Pediatrics, University College of Medical Sciences and Guru Tegh Bahadur Hospital, Dilshad Garden, Delhi. Ethical clearance was obtained from the hospital's ethical committee before the study commenced. Written informed consent was obtained from parents before disclosing the patient's course of hospital stay. Three cases of extubation failures that occurred in the PICU are discussed below.

2.1 Case 1

This case was a 19-month-old female who was admitted for pneumonia. Still, she later revealed a neuromuscular weakness due to spinal muscular atrophy, which led to multiple attempts of failed extubation, eventually leading to death. The details of the case are given in Table 1.

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Table 1: The details of case 1 with extubation failure.

Case 1		
Patient specifics	19 months, female	
Presenting complaints	Fever, cough, fast breathing for 10 days	
Other significant history	Reduced movements, gross motor development delay	
Examination findings at admission	Consciousness	Alert
	Heart Rate	116/min
	Respiratory Rate	46/min, with paradoxical breathing, pectus excavatum, bilateral wheeze, and crepitus
	Oxygen Saturation	72% on room air and 94% (Non-rebreathing mask)
	Axillary Temperature	99°F
	Capillary Refill Time	< 3sec
	Peripheral Pulses	good volume
	Generalized hypotonia with intact sensorium	
Provisional diagnosis	Bronchopneumonia with hypotonia under evaluation	
	Differential diagnosis- SMA type I/II/ congenital myopathy	
Course	Day1	Intravenous antibiotics, fluids, bronchodilators, adrenaline nebulization, and oxygen therapy with a non-rebreathing mask.
	Day 2	Respiratory distress worsened, moved to PICU, Intubated, and ventilated.
	Day 5	Sedation was tapered and stopped, and dexamethasone was started.
	Day 6 and 14	Extubation failure (respiratory acidosis and CO_2 retention) despite initial improvement
	Day 30	tracheostomy (delayed consent by attendants)
	Day 35	death
Extubation failure	Days 6 and 14	
Blood investigations	Hb- 8.5 gm/dl (anemia) TLC- 15600 mm^3 Platelets - 2.3 lakh/ mm^3 Blood Calcium level: 8.2mg/dl (Hypocalcemia) Vitamin D- 25.65ng/ml (Deficiency) Phosphate - 3.2 mg/dl(Low) Alkaline phosphatase- 210U/L (Elevated)	
	Kidney and liver functions- normal	
	Chest X-ray- right upper zone opacity	
	Genetic analysis- homozygous deletion in Exon 7 of the SMN1 gene	
Cultures	Blood- Staphylococcus aureus	
Final diagnosis	Pneumonia with Vitamin D deficiency, Rickets, with Spinal Muscular Atrophy Type 1, staphylococcal sepsis	

2.2 Case 2

This was a newborn who was admitted for hypoxic ischemic encephalopathy (HIE) stage III and intubated due to poor respiratory efforts. Prolonged ventilation resulted in diaphragmatic dysfunction, leading to extubation failure followed by death. The details of this case is explained in Table 2.

Table 2: The details of case 2 with extubation failure.

Case 2		
Patient specifics	Term, newborn female, birth weight 3.2 Kg	
Presenting complaints	Delayed cry and poor respiratory efforts	
Other significant history	Antenatal- uneventful, Natal- emergency lower segment cesarean section was done for non-progress of labor	
	Ventilated at birth, given intravenous fluids, antibiotics, and anticonvulsants for refractory seizures	
Examination findings at admission	Consciousness	Alert
	Heart Rate	148/min
	Respiratory Rate	Ventilator supported (no spontaneous breathing)
	Oxygen Saturation	98% on 70% FiO_2
	Axillary Temperature	37.1°C
	Capillary Refill Time	> 3sec
	Peripheral Pulses	Low volume
	Generalized hypotonia, sluggish reactive pupils of normal size, anterior fontanelle open and at the level, absent suck and Moro's reflexes	
Provisional diagnosis	Hypoxic ischemic encephalopathy(HIE III)	
Course	Day 1	Intubated, fluids, inotropes started, antibiotics started
	Day 7	Off inotropes and minimal FiO_2 requirements (no spontaneous respiratory efforts), antibiotics stopped
	Day 8	Thrombocytopenia, shock, inotropes, and empirical antifungal therapy were started after sending blood and urine investigations, and later started on targeted antifungal therapy
	Day 13	Dexamethasone started
	Day 14	Extubation failure due to poor efforts and respiratory acidosis, antibiotics upgraded according to Klebsiella positive tracheal aspirate, dexamethasone stopped after 48 hours
	Day 24	Dexamethasone restarted
	Day 25	Inotropes tapered, but ventilator requirement persisted, and extubation failed
	Day 58	Death due to progressive respiratory failure. Tracheostomy was not done due to negative consent
Extubation failure	Days 14 and 25	
Blood investigations	Initial complete blood counts, electrolytes, kidney and liver function test were normal	
	Ultrasound cranium- dilated ventricles	
	Ultrasound diaphragm- diaphragmatic atrophy and dysfunction, thickness fraction of 18%	
Cultures	Initial blood culture- sterile, Later blood culture- Candida glabrata	
	Tracheal aspirate- Klebsiella pneumoniae	

Final diagnosis	Hypoxic ischemic encephalopathy (HIE III), ventilator-associated pneumonia, Candida glabrata sepsis, and diaphragmatic dysfunction

2.3 Case 3

This was a child with tubercular meningitis, where extensive damage to the central nervous system due to disease pathology led to respiratory failure and intubation. Treatment in this patient could not reverse the irreversible brain damage already done due to the diseasecondition, resulting in failed attempts at extubation. This case is discussed in detail in Table 3.

Table 3: The details of Case 3 with extubation failure

Case 3		
Patient specifics	10 years, Male	
Presenting complaints	Fever for one and a half months and altered sensorium for 12 days	
Other significant history	Oral antibiotics on OPD basis	
Examination findings at admission	Consciousness	Alert
	Heart Rate	110/min
	Respiratory Rate	24/min
	Oxygen Saturation	87% on room air and 96% on oxygen mask
	Axillary Temperature	100°F
	Capillary Refill Time	< 3sec
	Peripheral Pulses	Palpable and good volume
	Generalized hypertonia, brisk reflexes, extensor plantar, positive meningeal signs, cranial nerves normal, no cerebellar dysfunction	
Provisional diagnosis	Subacute meningoencephalitis. Differential diagnosis: tubercular/ partially treated pyogenic meningitis	
Course	Day 1	Intubated in the emergency department for poor GCS, and started on antibiotics, hypertonic saline therapy. Generalized tonic-clonic seizures- started on antiepileptics and transferred to PICU
	Day 4	Started on antitubercular therapy with dexamethasone, as CECT head was suggestive of tubercular etiology
	Day 6	Partial improvement in tone, seizures controlled, GCS still poor, sedation tapered
	Day 7 and 15	Extubation failure.
	Day 15	Tracheostomy was done due to prolonged ventilatory requirement. The child became ventilator dependent, brainstem reflexes absent, developed hypertension, bed sores, urinary tract infection, antitubercular drug-induced hepatitis, and ventilator-associated pneumonia with left-sided empyema. Supportive and disease-appropriate treatment was given
Extubation failure	Days 7 and 15	
Blood investigations	Blood investigations were normal except for; Total leukocyte count(TLC) – 16500/ mm³.	
	Cerebrospinal fluid (CSF) examination: Protein levels- 117mg/dl Glucose- 80 mg/dl Microscopy revealed- 200 cells(70%lymphocytes)	
	CSF Catridge- based nucleic acid amplification test (CBNAAT) – negative	
	Contrast-enhanced CT brain- meningeal enhancement and ventriculomegaly, suggestive of tubercular meningitis	
Cultures	CSF culture- sterile	
Final diagnosis	Tubercular meningitis stage 3 with respiratory failure	

DISCUSSION

The requirement of mechanical ventilation is one of the major indications for PICU admissions. However, prolonged ventilation has its side effects, and its excessive usage can further add to the morbidity of patients. Therefore, it becomes crucial to discontinue ventilator support as soon as the patient shows improvement from their existing illness. Clinical impression alone cannot be a single criterion for deciding the ideal time to interrupt mechanical ventilation and give an extubation trial. Several key factors have been identified in the literature as major predictors of successful extubation. These include the primary disorder, younger age group, utilization of corticosteroids, application of non-invasive ventilation (NIV), and an extended duration of invasive mechanical ventilation (IMV) [3]. Neuromuscular conduction plays an important role, which may be affected by sedation, ventilatory muscle endurance, and ventilatory changes, such as increased physiological dead space, ventilatory muscle strength, and changes in ventilatory mechanics [4].

Sedation complicates weaning and extubation. Sedation is important in intubated patients for providing amnesia, reducing anxiety and stress response, and so improving tolerance to invasive ventilation, but studies have also shown an associated increased rate of extubation failure due to depressed central respiratory drive. Therefore, sedation in these patients should be monitored regularly by using validated and standardized tools like the state behavioral scale (SBS). A target SBS of 1(responsive to gentle touch or voice) and 0 (awake and able to calm) should be maintained in intubated pediatric patients. Levels of sedation should be kept as low as possible, especially during weaning [5, 6].

Respiratory muscle weakness is frequently seen in ICU patients and is associated with adverse outcomes. Infrequent

monitoring of respiratory muscle strength in these critically ill patients leads to missed detection of respiratory muscle weakness. The diaphragm is the major respiratory muscle responsible for spontaneous respiration; its dysfunction has now been identified as one of the causes of extubation failure. Ventilator-induced lung injury (VILI) and ventilator-induced diaphragmatic dysfunction (VIDD) are major causes affecting spontaneous respiration during extubation. Volutrauma and atelectrauma are two key mechanisms underlying VILI. Ventilator-induced diaphragmatic dysfunction is associated with weaning time, weaning outcome, ICU mortality, and long-term clinical outcomes.

Diaphragm ultrasonography is a non-invasive and easy-to-perform bedside test that has emerged as a promising technique for the evaluation of diaphragm contractility and morphology. It is a safe, painless, radiation-free, and cost-effective technique. It can easily be performed in ICU-admitted pediatric patients. Diaphragm thickness, excursion, and thickening fraction (DTf) are the three major ultrasound parameters that are identified to detect diaphragm dysfunction/paralysis, the change in the diaphragm, and guide predicting weaning and extubation success. A decrease in diaphragm thickness from baseline to nadir of ≥ 10% is considered diaphragm atrophy, while a diaphragmatic excursion of <10-14mm is considered significant. DTf of 30% – 36% is predictive of extubation failure [7, 8].

Controlled mechanical ventilation and high-pressure support ventilation should be avoided or weaned off early. Inspiratory muscle training, spontaneous breathing, continuous positive airway pressure (CPAP), use of lower ventilator driving pressures, and partially assisted modes of ventilation like neurally adjusted ventilatory assist (NAVA) or adaptive support ventilation (ASV) should be considered. Drugs like methylxanthines, i.e., theophylline and aminophylline, calcium sensitizers like levosimendan might help prevent VIDD. Temporary diaphragm pacing via transvenous or intramuscular route is also one experimental modality under trial to treat VIDD [9].

Recent studies indicate the role of non-invasive ventilation in treating post-extubation respiratory failures, which includes oxygen delivery modalities like support with oxygen via blow-by or aerosol mask, nasal cannula, high flow nasal cannula (HFNC), and non-invasive positive pressure ventilation (NIPPV), including CPAP and BiPAP. The role of NIPPV is limited in neurological causes [10, 11]. Usage of neuromuscular blockers, aminoglycosides, and high-dose corticosteroids, sepsis, metabolic derangements, prolonged hyperglycemia, and malnutrition lead to mitochondrial oxidative stress and inflammation in these patients and contribute to the risk of extubation failure [9].

A strategic weaning plan is required for successful extubation in PICUs. Corticosteroids like dexamethasone to prevent and treat post-extubation stridor have been used in high-risk children or neonates. Disease reversibility and chronicity should also be considered when deciding extubation readiness, as patients with rapidly reversible respiratory disease wean quickly and extubate faster than patients with slowly reversible and chronic issues. A good nutritional status should be maintained to promote successful extubation, as it reduces diaphragmatic atrophy and dysfunction in critically ill children who are mechanically ventilated. Intensivists can use tests, like extubation readiness testing, leak test, and spontaneous breathing trials, to guide extubation. Many indices have been defined in the literature and can be used along with clinical assessment to predict the success of extubation. Some examples are spontaneous ventilatory frequency, Spontaneous tidal volume, Rapid Shallow Breathing Index (RSBI), Compliance, Resistance, Oxygenation, Pressure (CROP) index, Volumetric capnography, and crying vital capacity (CVC) [12].

CONCLUSION

The extubation process is not a singular event but a meticulously planned and gradual procedure. It involves a multitude of factors that significantly influence its success. Prioritizing extubation preparation becomes the second most important consideration following the resolution of the primary disorder. This preparatory phase should ideally commence from the very onset of intubation, emphasizing continuous monitoring and proactive measures to prevent respiratory muscle damage. By integrating thoughtful planning and vigilant monitoring, the extubation process can be optimized, ensuring a smoother transition and promoting positive patient outcomes.

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