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Bronchoscopic Resolution of Refractory Atelectasis in a Toddler with Polymicrobial MDR Pneumonia: A Case Report

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A B S T R A C T

Pediatric acute respiratory distress syndrome (PARDS) complicated by ventilator-associated pneumonia (VAP) poses significant management challenges, particularly when caused by multidrug-resistant organisms such as *Stenotrophomonas maltophilia* and *Pseudomonas aeruginosa*. A frequent and deleterious complication is plate-like atelectasis, which may prove refractory to conservative management due to anatomical constraints in the pediatric airway and biofilm formation. A 23-month-old male presented with severe PARDS and polymicrobial VAP. Despite extubation to High-Flow Nasal Cannula (HFNC), the patient developed persistent right upper lobe plate-like atelectasis refractory to aggressive physiotherapy and targeted antibiotic therapy with Levofloxacin and Ceftazidime for 21 days. On Day 75 of illness, a flexible bronchoscopy was performed. Intraoperative findings revealed hyperemic mucosa without macroscopic mucus plugging. However, the procedure, involving saline lavage and suctioning, resulted in immediate recruitment. Within 24 hours, the respiratory rate decreased from 45 to 24 breaths per minute, and the SpO_2/FiO_2 ratio improved significantly from 185 to 310, allowing weaning from respiratory support. In conclusion, in toddlers with multidrug-resistant VAP, atelectasis may persist due to biofilm-mediated micro-obstruction rather than macroscopic plugging. Flexible bronchoscopy is a safe and effective therapeutic adjunct in these cases, facilitating distal airway recruitment and breaking the cycle of chronic infection.

1. Introduction

Acute respiratory distress syndrome (ARDS) in the pediatric population constitutes a life-threatening pathology characterized by severe hypoxemia, reduced lung compliance, and diffuse alveolar damage.[1] A frequent and deleterious complication of ARDS is ventilator-associated pneumonia (VAP), which affects a significant proportion of mechanically ventilated pediatric patients.[2] The synergy between ARDS and VAP creates a vicious pathological cycle; the inflammatory exudate and loss of surfactant function inherent to ARDS predispose the lung parenchyma to bacterial colonization, while VAP further inactivates surfactant and promotes alveolar collapse, clinically manifesting as atelectasis.[3]

In the pediatric airway, anatomical factors significantly increase the risk of atelectasis compared to the adult population. These factors include smaller airway diameters, which increase resistance inversely to the fourth power of the radius, fewer collateral ventilation channels, such as the pores of Kohn and canals of Lambert, and a highly compliant chest wall that offers less outward recoil to counterbalance alveolar collapse.[4] Consequently, once a pediatric lung unit collapses, re-expansion requires significantly higher opening pressures, often exceeding safe

ventilation limits.[5]

Plate-like atelectasis, a subtype characterized by linear collapse often perpendicular to the pleural surface, is frequently associated with poor regional ventilation and persistent infection.[6] When this condition becomes refractory to conservative management strategies, including chest physiotherapy, positioning, hypertonic saline nebulization, and recruitment maneuvers, it severely compromises oxygenation and delays recovery.

The microbiological landscape further complicates clinical management. *Stenotrophomonas maltophilia* is an emerging multidrug-resistant Gram-negative bacillus associated with high mortality rates in immunocompromised hosts and prolonged Intensive Care Unit stays.[7] Co-infection with *Pseudomonas aeruginosa* indicates a complex biofilm-associated pathology that is notoriously difficult to eradicate. These pathogens are known to produce robust alginate and extracellular matrix biofilms that line the distal airways, increasing surface tension and predisposing the lung to adhesive atelectasis even in the absence of macroscopic obstruction.[8]

While flexible bronchoscopy is an established intervention for airway clearance in lobar collapse due to visible mucus plugging, its utility in plate-like atelectasis, where the obstruction may be distal or adhesive, is less defined in current guidelines. Current pediatric literature regarding the efficacy of bronchoscopy in toddlers with findings of a clean airway without macroscopic plugs remains scarce.[9,10]

This study aims to report the clinical trajectory and successful management of a toddler with severe ARDS and polymicrobial VAP caused by *Stenotrophomonas* and *Pseudomonas* complicated by refractory plate-like atelectasis. The novelty of this report lies in demonstrating the therapeutic value of flexible bronchoscopy in a case where radiographic opacity persisted despite the absence of macroscopic mucus plugs, offering insights into the pathophysiological benefits of distal airway clearance and biofilm disruption in multidrug-resistant infections.

2. Case Presentation

Written informed consent was obtained from the parents of the patient for the procedure, anesthesia, and the publication of medical data including radiographic images.

The clinical narrative centers on a 23-month-old male of Balinese ethnicity, weighing 11 kg, who presented to the Pediatric Intensive Care Unit (PICU) on October 20th, 2023. This date, designated as Day 1 of the clinical timeline, marked the culmination of a week-long prodrome characterized by progressive and unrelenting dyspnea. Upon admission, the patient's clinical presentation was consistent with severe respiratory compromise; physical examination revealed significant work of breathing, manifested by visible suprasternal and intercostal retractions, alongside audible coarse crackles and retained secretions indicative of mucociliary clearance failure. Notably, the patient's medical history was unremarkable, with no documented antecedents of reactive airway disease, asthma, foreign body aspiration, or congenital immunodeficiency, suggesting that the severity of the presentation was driven by an acute, high-burden infectious insult rather than a chronic underlying pathology (Table 1).

The patient's hospital course was protracted, spanning over two months. Retrospective analysis of the clinical data reveals a trajectory defined by three distinct pathophysiological phases: acute decompensation, complex nosocomial superinfection, and a prolonged period of refractory sequelae. The initial phase of management was characterized by a struggle to stabilize gas exchange amidst worsening lung compliance. Initial therapeutic strategies focused on the management of severe pneumonia utilizing non-invasive respiratory support.

Table 1. Summary of Clinical Findings on Admission

Patient Demographics	**23-month-old Male** Ethnicity: Balinese Weight: 11 kg
Chief Complaint	**Severe Dyspnea** Onset: Progressive worsening over the preceding 7 days
Respiratory Inspection	Signs of significant work of breathing: • **Suprasternal Retractions** (Visible) • **Intercostal Retractions** (Visible)
Auscultation Findings	• **Coarse Crackles** (Audible) • Signs of retained secretions (Mucociliary clearance failure)
Past Medical History	• Asthma: Negative • Foreign Body Aspiration: Negative • Immunodeficiency: Negative
Initial Assessment	Severe Pneumonia with acute respiratory compromise. *(Prior to progression to Pediatric ARDS on Day 22)*

However, these measures proved insufficient as the patient exhibited signs of increasing respiratory muscle fatigue and worsening hypoxemia. The clinical inflection point occurred on November 11th, 2023 (Day 22). On this date, arterial blood gas analysis revealed severe oxygenation failure with a PaO_2/FiO_2 ratio dropping below 100 mmHg. This met the Berlin criteria and PALICC (Pediatric Acute Lung Injury Consensus Conference) definitions for severe pediatric acute respiratory distress syndrome (PARDS). Consequently, the decision was made to escalate care to invasive mechanical ventilation to facilitate lung protective strategies, optimize positive end-expiratory pressure (PEEP), and reduce the metabolic cost of breathing.

Following stabilization on mechanical ventilation, the second phase of the clinical course was complicated by nosocomial superinfection. After two weeks of invasive ventilation, the patient developed new febrile episodes accompanied by a qualitative change in tracheal secretions, which became purulent and copious. This clinical picture raised immediate suspicion for ventilator-associated pneumonia (VAP).

On day 36, tracheal aspirate cultures provided a definitive microbiological diagnosis, revealing a complex polymicrobial coinfection involving two distinct multidrug-resistant (MDR) Gram-negative pathogens: (1) *Stenotrophomonas maltophilia:* This organism presented a significant therapeutic challenge due to its intrinsic resistance mechanisms.

The antibiogram confirmed resistance to Carbapenems and Aminoglycosides—classes often used empirically in PICU settings. Crucially, the isolate retained sensitivity to Levofloxacin and Trimethoprim-Sulfamethoxazole; (2) *Pseudomonas aeruginosa:* Concurrent isolation of this pathogen revealed a multidrug-resistant phenotype, though it remained susceptible to Ceftazidime and Amikacin.

The identification of these pathogens explained the lack of clinical response to the initial broad-spectrum coverage with Meropenem, as *Stenotrophomonas* is intrinsically resistant to carbapenems via the production of metallo-beta-lactamases. Guided by pharmacodynamic principles and the specific sensitivity patterns identified, the medical team initiated a targeted, 21-day combination antimicrobial regimen. This consisted of Levofloxacin (dosed at 10 mg/kg intravenously every 12 hours) to target the *Stenotrophomonas*, and Ceftazidime (dosed at 50 mg/kg intravenously every 8 hours) to address the *Pseudomonas* burden. This tailored approach was critical in sterilizing the airway and resolving the systemic inflammatory response.

The third and most prolonged phase of the clinical course began on Day 53. Following the successful resolution of systemic sepsis markers and improvement in lung compliance, the patient was extubated to high-flow nasal cannula (HFNC). While the extubation was successful in terms of airway patency, immediate post-extubation radiography revealed a new complication: a dense, plate-like atelectasis located in the right upper lobe (RUL). The management of this atelectasis represented a clinical dilemma. Given the patient's recent recovery from severe PARDS, the primary goal was to avoid re-intubation. Therefore, a stepwise escalation protocol was adopted, prioritizing non-invasive clearance methods over a period of three weeks. (i) Weeks 1-2 (Conservative Clearance Trial): The initial strategy involved aggressive chest physiotherapy, including mechanical percussion and vibration delivered every four hours to shear mucus from the airway walls. This was augmented by the administration of nebulized 3% hypertonic saline, intended to create an osmotic gradient in the airway aimed at rehydrating the mucus layer and reducing viscosity. Additionally, the patient was placed in the left lateral decubitus position to utilize gravity to facilitate drainage of the affected right upper lobe; (ii) Week 3 (Definition of Refractory Status): Despite strict adherence to this multimodal conservative regimen, serial imaging showed no improvement in the RUL opacity. Clinically, the patient remained tachypneic with labile oxygen saturation, which prevented any attempt to wean the HFNC settings. This 21-day plateau of unresponsiveness to optimal non-invasive therapy formally defined the condition as refractory atelectasis, necessitating a re-evaluation of the therapeutic approach (Table 2).

On day 74, the patient remained alert but exhibited persistent respiratory compromise. Assessment on Day 74 provided the crucial data points that justified invasive intervention. The patient demonstrated tachypnea with a respiratory rate of 45 breaths/minute. Oxygenation remained fragile, with SpO_2 fluctuating between 92% and 93% despite substantial support via HFNC (FiO_2 35% at a flow rate of 15 L/min). Examination of the thorax revealed asymmetrical chest expansion with a marked reduction on the right side. Auscultation corroborated the radiological findings, demonstrating diminished air entry in the right upper zone accompanied by bilateral coarse crackles, suggesting both focal collapse and diffuse secretory burden. A pivotal finding in the pre-procedural workup was the thyroid function profile, which indicated low T3 and T4 levels with a normal TSH. This pattern is pathognomonic for Euthyroid Sick Syndrome (Non-thyroidal Illness Syndrome), a maladaptive response to chronic critical illness. The clinical relevance of this finding cannot be overstated; the associated hypothyroid state likely contributed to generalized myopathy and respiratory muscle wasting. This metabolic weakness offered a

mechanistic explanation for the failure of the conservative physiotherapy trial: the patient simply lacked the muscular strength to generate an effective cough to clear the RUL obstruction. The Chest X-ray confirmed the persistence of the RUL linear opacity. Lung ultrasound was utilized to further characterize the lesion, revealing B-lines and subpleural consolidation. Crucially, the ultrasound excluded significant pleural effusion, confirming that the etiology was intraluminal or parenchymal (and thus potentially amenable to bronchoscopy) rather than compressive.

On day 75, faced with the failure of the 3-week conservative trial and the metabolic evidence of poor cough mechanics, the multidisciplinary team proceeded with a diagnostic and therapeutic flexible bronchoscopy. Safety was paramount given the patient's fragile respiratory reserve. The procedure was performed under general anesthesia utilizing a Sevoflurane and Propofol induction. A size 2.0 laryngeal mask airway (LMA) was selected as the conduit for the bronchoscope. This decision was strategic; unlike an endotracheal tube, the LMA minimized airway resistance and allowed the bronchoscopist to perform a dynamic assessment of the trachea and carina without the visual obstruction of a tube tip. Detailed visual inspection of the tracheobronchial tree revealed normal anatomy without evidence of tracheomalacia or external compression. The mucosa of the right upper lobe was noted to be significantly hyperemic and edematous, consistent with the chronic inflammatory milieu of the preceding weeks. The most significant intraoperative finding was the absence of macroscopic mucus plugs. The segmental bronchi appeared patent to visual inspection. This finding challenged the traditional assumption that atelectasis is solely caused by large, obstructing plugs. Despite the clean macroscopic appearance, the team proceeded with bronchoalveolar lavage (BAL) utilizing 30 ml of warm 0.9% saline. Suctioning was applied to the subsegmental bronchi. This maneuver resulted in the retrieval of turbid fluid, confirming the hypothesis that the obstruction was located distally—likely composed of biofilm and micro-secretions entrenched in the smaller airways (generations 8-12), which are non-visible to the naked eye but responsive to the hydraulic and mechanical forces of lavage and suction.

The clinical impact of the bronchoscopic intervention was profound and immediate, validating the concept of micro-recruitment. Within 24 hours post-procedure, the patient's respiratory mechanics underwent a dramatic normalization. The respiratory rate dropped from a tachypneic 45 breaths/minute to a physiologic 24 breaths/minute. Gas exchange efficiency improved substantially, with the SpO_2/FiO_2 ratio jumping from 185 to 310, indicating successful alveolar recruitment and reduction of intrapulmonary shunting. This physiological improvement was mirrored by radiological evidence, with follow-up imaging confirming aeration of the previously collapsed right upper lobe. The rapid resolution allowed for the swift weaning of respiratory support; the patient was transitioned to room air on January 5th, 2024, and was deemed fit for discharge from the PICU just two days later, marking the conclusion of a complex and challenging critical care journey.

3. Discussion

The clinical trajectory of this case presents a compelling paradox that challenges the foundational dogma of pediatric bronchoscopy. Traditionally, the therapeutic utility of flexible bronchoscopy in the context of atelectasis is predicated on the plumbing model—the visualization and physical removal of a macroscopic obstruction, typically a dense mucus plug or a foreign body. Under this paradigm, the absence of visible obstruction is often interpreted as a negative study, leading to the assumption that the procedure yielded no therapeutic benefit.[11]

Table 2. Diagnosis, Treatment, Follow-up, and Outcome

I. FINAL DIAGNOSIS PROFILE	
Primary Pulmonary Diagnosis	**1. Severe Pediatric ARDS** (Resolved Phase) **2. Polymicrobial VAP** (MDR Organisms): • **Stenotrophomonas maltophilia** (Carbapenem Resistant) • **Pseudomonas aeruginosa** (MDR Phenotype)
Complications & Co-morbidities	• **Refractory Plate-like Atelectasis** (Right Upper Lobe) • **Euthyroid Sick Syndrome** (Low T3/T4, Normal TSH)
II. THERAPEUTIC MANAGEMENT	
Targeted Pharmacotherapy **(21-Day Regimen)**	*Regimen guided by specific sensitivity patterns:* • `Levofloxacin` 10 mg/kg IV q12h (Targeting *S. maltophilia*) • `Ceftazidime` 50 mg/kg IV q8h (Targeting *P. aeruginosa*)
Conservative Measures **(Failed Trial: Days 53-74)**	• High-Flow Nasal Cannula (HFNC) Support • Chest Physiotherapy (Percussion/Vibration q4h) • Nebulized 3% Hypertonic Saline • Positional Therapy (Left Lateral Decubitus)
Interventional Procedure	**Flexible Bronchoscopy under General Anesthesia** • **Airway:** Laryngeal Mask Airway (LMA) Size 2.0 • **Technique:** Bronchoalveolar Lavage (30 ml warm saline) + Distal Suctioning • **Finding:** Hyperemic mucosa, **No macroscopic mucus plugs**
III. FOLLOW-UP & CLINICAL OUTCOME	
Immediate Post-Procedure **(24 Hours)**	**Respiratory Rate:** Improved from 45 → **24 breaths/min** **Gas Exchange (P/F Ratio):** Improved from 185 → **310** **Radiology:** Significant aeration of Right Upper Lobe
Final Disposition	• **Weaning:** Transitioned to room air on Jan 5, 2024 • **Discharge:** Discharged from PICU on Jan 7, 2024 • **Full clinical resolution of atelectasis**

However, our patient demonstrated a stark dichotomy between the endoscopic findings and the clinical outcome: the airways appeared macroscopically patent and free of large plugs, yet the right upper lobe atelectasis, which had been refractory to three weeks of aggressive conservative management, resolved almost immediately following the procedure. This phenomenon necessitates a shift

in our pathophysiological understanding from macroscopic obstruction to microscopic adhesion. The persistence of atelectasis in a clean airway suggests that the pathology was not located in the central bronchi (generations 1–4), which are visible to the bronchoscope, but rather in the distal bronchioles (generations 8–12). In this distinct anatomical niche, the mechanism of collapse is likely not physical occlusion by a solid mass, but rather adhesive atelectasis driven by alterations in surface tension and airway resistance (Figure 1).[12]

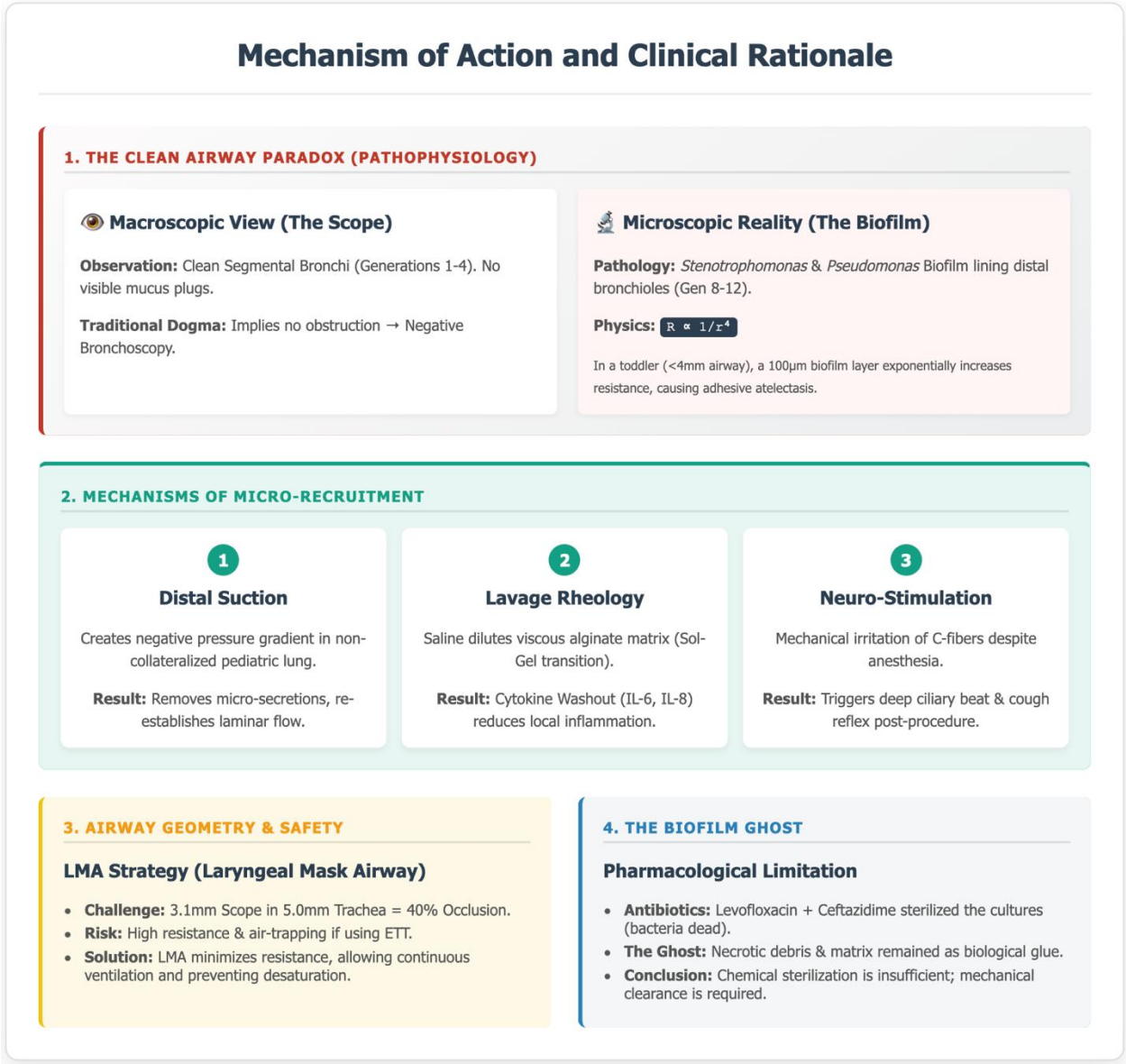

Figure 1. Mechanism of action and clinical rationale of this study.

The primary driver of this adhesive pathology in our patient was the synergistic coinfection of *Stenotrophomonas maltophilia* and *Pseudomonas aeruginosa*. These organisms are not merely planktonic bacteria; they are sophisticated biofilm engineers. *Pseudomonas aeruginosa* is renowned for

its overproduction of alginate, an exopolysaccharide that forms a viscous, protective matrix.[13] *Stenotrophomonas maltophilia*, while less capable of initiating biofilm formation on its own, possesses a unique ability to adhere to and colonize the pre-formed alginate matrices of *Pseudomonas*. This co-colonization creates a super-biofilm that is mechanically robust and highly resistant to clearance. In the pediatric lung, the implications of this biofilm lining are governed by the laws of physics, specifically Poiseuille's Law and Laplace's Law. A toddler's distal airway may have a diameter of less than 4 mm. In an adult airway of 15 mm, a biofilm layer of 100 microns is negligible. However, in a pediatric airway, that same 100-micron layer significantly reduces the luminal radius. According to Poiseuille's Law, resistance to airflow is inversely proportional to the radius to the fourth power ($R\alpha 1/r^4$).[14] Therefore, even a microscopic reduction in radius caused by a biofilm lining causes an exponential increase in airway resistance. Furthermore, the biofilm alters the surface tension of the airway lining fluid, increasing the opening pressure required to inflate the alveoli (LaPlace's Law). We hypothesize that these biofilms acted as a biological glue, sealing the distal airways and preventing recruitment despite high-flow support, a pathology completely invisible to the macroscopic view of the bronchoscope.[15]

The immediate resolution of atelectasis in this case supports the concept of micro-recruitment. The therapeutic efficacy of bronchoscopy in this context was not derived from the extraction of a single large plug, but rather from the cumulative physiological effects of the procedure itself.[16] We propose a threefold mechanism of action that explains how navigating a clean airway resulted in significant clinical improvement. The application of suction during bronchoscopy does more than simply remove fluid; it creates a dynamic pressure gradient. When the tip of the bronchoscope is wedged into a segmental bronchus and suction is applied, it generates localized negative pressure. In the pediatric lung, which lacks significant collateral ventilation due to poorly developed pores of Kohn, this negative pressure can paradoxically assist in recruitment. By clearing the micro-secretions and biofilm debris from the patent segment, the suction reduces the critical opening pressure of the distal units. Furthermore, the removal of the biofilm burden decreases the turbulent flow within the small airways, re-establishing laminar airflow to the atelectatic segments. This allows the positive pressure from the High-Flow Nasal Cannula (HFNC) to finally reach the alveoli effectively post-procedure.[17]

The administration of warm saline lavage served a crucial rheological function. The biofilm matrix produced by *Pseudomonas* and *Stenotrophomonas* is highly viscous and thixotropic—it becomes more fluid when agitated but solidifies when static. The static nature of the atelectatic lobe allowed the biofilm to harden into a cement-like layer. The introduction of saline diluted the concentration of the exopolysaccharides, transitioning the biofilm from a gel phase to a sol (liquid) phase. Additionally, the lavage likely performed a cytokine washout. Atelectatic lung segments act as reservoirs for inflammatory mediators. In chronic VAP, the distal airways are saturated with Interleukin-6 (IL-6), Interleukin-8 (IL-8), and tumor necrosis factor-alpha (TNF-).[18] These cytokines perpetuate inflammation, causing localized edema and surfactant inactivation. By physically washing out this inflammatory soup, the lavage reduced the local oncotic pressure and dampened the inflammatory cascade that was perpetuating alveolar collapse.

Perhaps the most overlooked mechanism is the stimulation of the patient's intrinsic clearance systems. The patient had been suffering from Euthyroid Sick Syndrome and chronic critical illness myopathy, leading to a weak, ineffective cough. Despite the use of general anesthesia, the mechanical presence of the bronchoscope within the

tracheobronchial tree stimulates deep sensory receptors (rapidly adapting receptors and C-fibers). This irritation triggers a profound, albeit suppressed, cough reflex and stimulates ciliary beat frequency. Post-procedure, as the anesthesia wears off, this heightened state of ciliary activation persists, allowing the patient to mobilize secretions from the periphery to the central airways more effectively than chest physiotherapy alone could achieve.

The successful management of this case relied heavily on navigating the pharmacological minefield of multidrug-resistant (MDR) Gram-negative coinfection. *Stenotrophomonas maltophilia* represents a formidable therapeutic challenge in the pediatric ICU. Unlike most Gram-negative bacilli, it possesses intrinsic resistance to carbapenems due to the production of two inducible chromosomal beta-lactamases: L1 (a metallo-beta-lactamase) and L2 (a serine cephalosporinase). This renders the standard escalation antibiotic, Meropenem, completely ineffective, and in some cases, counterproductive, as it induces these enzymes.[19]

The selection of Levofloxacin and Ceftazidime in this case warrants specific discussion. While Trimethoprim-Sulfamethoxazole (TMP-SMX) is traditionally considered the drug of choice for *Stenotrophomonas*, its use in critically ill toddlers can be limited by potential hematological toxicity, hypersensitivity reactions, or dosing difficulties in patients with fluid restrictions. Levofloxacin emerged as a vital alternative in this protocol. As a fluoroquinolone, it exhibits excellent bioavailability and, crucially, high tissue penetration into the lung parenchyma and epithelial lining fluid—concentrations that often exceed serum levels. This property is essential when targeting biofilm-associated infections where antibiotic diffusion is physically impeded.

Concurrently, the *Pseudomonas aeruginosa* isolate displayed an MDR phenotype but retained sensitivity to Ceftazidime. The decision to use combination therapy was strategic, aiming to exploit potential synergy and prevent the emergence of further resistance during the prolonged 21-day course. However, the persistence of atelectasis despite the microbiological sterilization of the airways highlights a critical limitation of antimicrobial therapy: The biofilm ghost phenomenon. Antibiotics are chemical agents; they kill bacteria, but they do not disintegrate the physical structure of the biofilm matrix or the cellular debris left behind. Even after the bacteria were eradicated, the alginate matrix and necrotic neutrophil debris likely remained lining the airways, maintaining the mechanical obstruction. This underscores that in biofilm-associated VAP, chemical sterilization is insufficient; mechanical clearance via bronchoscopy provides the necessary adjunct to remove the physical residue of the infection that antibiotics cannot dissolve.[20]

The decision to perform bronchoscopy in a 23-month-old with a history of severe ARDS is not trivial and requires a rigorous risk-benefit analysis centered on airway geometry. The pediatric airway is not merely a miniaturized adult airway; it is a highly vulnerable conduit where the margin for error is measured in millimeters. In a toddler, the trachea may have a diameter of approximately 5.0 mm. Introducing a flexible bronchoscope with an outer diameter of 3.1 mm significantly compromises the available cross-sectional area for ventilation. Mathematically, the area of a 5.0 mm airway is approximately 19.6 mm^2. A 3.1 mm scope occupies roughly 7.5 mm^2, occluding nearly 40% of the airway lumen. If performed through an endotracheal tube (ETT), the constraints are even more severe. An age-appropriate 4.5 mm ETT has an internal diameter that would be nearly entirely occluded by the scope, creating a high-resistance system that risks air-trapping, auto-PEEP generation, and barotrauma, alongside precipitous desaturation.

To mitigate these risks, our airway strategy utilized a laryngeal mask airway (LMA). This approach represents a paradigm of minimally invasive airway

control. By sitting in the hypopharynx rather than the trachea, the LMA allows the bronchoscope to enter the trachea directly. This preserves the potential space between the scope and the tracheal wall for gas exchange, significantly reducing resistance compared to the ETT approach. Furthermore, the LMA allows for continuous ventilation during the procedure. The absence of desaturation events or hemodynamic instability in our patient validates this strategy. It demonstrates that even in a lung recently recovering from ARDS, bronchoscopy is safe, provided that the geometry of ventilation is respected—utilizing the largest possible airway conduit (the LMA) for the necessary scope size.[17,18]

While the outcomes of this case are promising, the interpretation of the findings must be tempered by the inherent limitations of a single case report. The association between the bronchoscopic intervention and the resolution of atelectasis is temporally strong but lacks the control of a randomized trial. We cannot definitively rule out the possibility that the cumulative effect of the three-week antibiotic course reached a tipping point coincidentally at the time of the procedure, although the immediate physiological improvement suggests otherwise. From a pathological perspective, our biofilm theory remains a clinical hypothesis derived from the known behavior of the cultured pathogens. We did not perform electron microscopy or specific biofilm staining on the lavage fluid to visually confirm the presence of the alginate matrix. Future studies utilizing such diagnostic modalities would provide definitive proof of the micro-obstruction model. Furthermore, this case highlights a gap in current guidelines regarding the timing of intervention. The 21-day refractory period represents a significant duration of morbidity. Future research should focus on randomized controlled trials comparing early bronchoscopy (after 7 days of failed conservative therapy) versus late bronchoscopy (standard care). Identifying predictive biomarkers or radiographic signs that distinguish plug-mediated from biofilm-mediated atelectasis could help clinicians deploy this tool earlier in the disease course, potentially reducing PICU length of stay and ventilator days.[19,20]

4. Conclusion

The management of pediatric acute respiratory distress syndrome (PARDS) complicated by polymicrobial multidrug-resistant ventilator-associated pneumonia (VAP) remains one of the most formidable challenges in pediatric critical care. This case report illuminates a specific, often under-recognized sequela of this condition: refractory plate-like atelectasis driven by biofilm-producing organisms. The resolution of this patient's condition offers three critical takeaways for the clinician. First, it deconstructs the traditional dogma that flexible bronchoscopy is only indicated when macroscopic mucus plugging is suspected. We demonstrated that in the presence of biofilm-producing pathogens like *Stenotrophomonas maltophilia* and *Pseudomonas aeruginosa*, the obstruction is often microscopic, adhesive, and distal—invisible to the eye but amenable to mechanical disruption. The absence of visible plugs should not be interpreted as a failure of the procedure, nor should it deter the intensivist from considering bronchoscopy in patients with clean airways on imaging who fail to recruit. Second, this case validates the safety and efficacy of flexible bronchoscopy as a therapeutic adjunct in toddlers, provided that a rigorous airway strategy is employed. The use of a laryngeal mask airway (LMA) to mitigate resistance, combined with targeted lavage and suction, can break the cycle of chronic infection and collapse without compromising patient safety. Finally, this report underscores the limitations of antimicrobial therapy in isolation. While targeted antibiotics are essential for bacterial eradication, they cannot address the physical sequelae of the infection—the biofilm matrix and inflammatory debris that act as a biological glue. In such recalcitrant cases, flexible bronchoscopy acts not

merely as a diagnostic tool, but as a necessary mechanical synergist to pharmacological treatment. In conclusion, persistent atelectasis in the context of MDR VAP should prompt early consideration for bronchoscopic intervention, regardless of the radiographic absence of large plugs. By targeting the distal micro-environment, clinicians can facilitate lung recruitment, accelerate weaning, and improve outcomes in this vulnerable pediatric population. This proactive approach transitions bronchoscopy from a last resort rescue maneuver to an integral component of the comprehensive management strategy for complex pediatric pneumonia.

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