

Chest Physiotherapy as an Alternative to Bronchoscopy in Mucus Plug–Induced Atelectasis: A Case Report

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Abstract

Pneumonia in patients with neurological disorders is associated with impaired airway protection, ineffective cough, and increased risk of secretion retention, which may lead to complications such as mucus plug–induced atelectasis. This case report describes a 60-year-old female with a history of ischemic stroke, vascular dementia, and prolonged immobilization who presented with dyspnea, fever, and difficulty expectorating sputum. Initial evaluation supported a diagnosis of pneumonia, which subsequently progressed to respiratory failure, sepsis, and acute respiratory distress syndrome (ARDS). During hospitalization, the patient developed acute deterioration with radiographic findings suggestive of left-sided atelectasis caused by mucus plugging.

Due to limited availability of bronchoscopy, chest physiotherapy was implemented as the primary intervention for airway clearance. Management included a combination of suctioning, postural drainage, percussion, vibration, and positioning strategies, along with mechanical ventilation and supportive care. Gradual clinical improvement was observed, as evidenced by enhanced oxygenation, reduction in sputum retention, and radiological re-expansion of the affected lung. The patient was successfully weaned from mechanical ventilation, underwent tracheostomy due to persistent airway clearance impairment, and showed continued recovery.

This case highlights the important role of chest physiotherapy as a non-invasive and effective modality in managing mucus plug–induced atelectasis, particularly in patients with neurological impairment and in settings where bronchoscopy is not readily available. Early recognition of secretion retention and aggressive airway clearance strategies are essential to improve respiratory outcomes and prevent further complications in this high-risk population.

Keywords: Pneumonia, Atelectasis, Chest physiotherapy, Stroke, Respiratory failure

Introduction

Pneumonia remains a major cause of morbidity and mortality, particularly in elderly patients and those with neurological disorders. Aspiration pneumonia is common in this population due to impaired airway protection mechanisms. Cough and swallowing are essential defenses against aspiration; however, both are frequently compromised in neurological conditions such as stroke, increasing the risk of pneumonia.¹ In post-stroke patients, decreased mastication, salivation, swallowing function, and poor oral hygiene disrupt oral microbial balance, facilitating colonization by pathogenic bacteria that may be aspirated into

the lower respiratory tract. Additionally, post-stroke immunodeficiency further predisposes these patients to infection.¹

The burden of pneumonia in older adults extends beyond the acute phase, with high short- and long-term mortality. In-hospital mortality in elderly patients with community-acquired pneumonia reaches 20.4%, while one-year mortality can be as high as 54.9%, even among those discharged alive.² Aspiration pneumonia is associated with worse outcomes, including more severe neurological impairment and prolonged hospitalization, with an average increase of 10 days compared to non-aspiration cases.³ These findings underscore the significant clinical impact of pneumonia in this vulnerable population.

Impaired airway clearance further contributes to disease progression, leading to secretion retention, mucus plugging, and complications such as atelectasis, which exacerbate respiratory failure. Therefore, optimizing airway clearance is a key component of management. This case report highlights the role of chest physiotherapy as a non-invasive intervention in the management of mucus plug-induced atelectasis in a pneumonia patient with underlying neurological disorders.

Case report

A 60-year-old female patient presented to the hospital with chief complaints of dyspnea, fever, and difficulty expectorating sputum due to an ineffective cough, accompanied by decreased appetite for several days prior to admission. The patient had a history of ischemic stroke two years earlier, resulting in residual quadriparesis and aphasia, as well as vascular dementia and prolonged bedridden status.

On initial evaluation in the emergency department, the patient appeared moderately ill with a Glasgow Coma Scale (GCS) of E4VaphasiaM6, a body temperature of 38.1°C, tachypnea, and tachycardia. Initial lung auscultation revealed no crackles; however, suctioning yielded a large amount of whitish-yellow sputum. Laboratory investigations demonstrated leukocytosis with neutrophil predominance, impaired renal function, and hyperglycemia. Chest radiography showed an infiltrate in the right lower lung zone. Based on these findings, the patient was diagnosed with pneumonia in a geriatric patient with comorbid vascular dementia and post-stroke sequelae.

Initial management included airway and supportive care with oxygen administration via nasal cannula at 2 L/min, nasogastric tube (NGT) placement, and secretion suctioning. Pharmacological therapy consisted of intravenous ampicillin-sulbactam 1.5 g every 8 hours, intravenous fluids with Ringer’s lactate at 20 drops per minute, intravenous omeprazole 40 mg, N-acetylcysteine 1000 mg per 24 hours, intravenous citicoline 1 g every 12 hours, and paracetamol as needed with a target temperature below 36.9°C. The patient’s chronic medications (amlodipine, donepezil, risperidone, and clopidogrel) were continued. Sputum and blood cultures were obtained.

Table 1. Hematology laboratory findings

Parameter	Feb 17 th	Feb 19 th	Feb 23 rd	Feb 27 th	Mar 1 st	Reference Range
Hemoglobin (g/dL)	10.6 ↓	10.3 ↓	9.8 ↓	9.9 ↓	8.1 ↓	11.7 – 15.5
Hematocrit (%)	31.8 ↓	30.7 ↓	31.0 ↓	29.2 ↓	23.2 ↓	35 – 47
RBC (10 ⁶ /μL)	4.12	4.05	–	–	–	3.8 – 5.2
WBC (10 ³ /μL)	18.70 ↑	30.45 ↑	31.45 ↑	30.95 ↑	16.15 ↑	3.6 – 11.0
Platelets (10 ³ /μL)	404	319	404	452 ↑	598 ↑	150 – 440
Neutrophils (%)	90 ↑	95 ↑	–	–	–	50 – 70

Parameter	Feb 17 th	Feb 19 th	Feb 23 rd	Feb 27 th	Mar 1 st	Reference Range
Lymphocytes (%)	5 ↓	3 ↓	–	–	–	20 – 40
MCV (fL)	77.2 ↓	75.8 ↓	–	–	–	80 – 100
MCH (pg)	25.7 ↓	25.4 ↓	–	–	–	26 – 34
MCHC (g/dL)	33.3	33.6	–	–	–	32 – 36

Table 2. Clinical chemistry laboratory findings

Parameter	Feb 17 th	Feb 24 th	Feb 25 th	Feb 27 th	Mar 1 st	Reference Range
Urea (mg/dL)	59 ↑	41		35	18	21 – 43
Creatinine (mg/dL)	0.97 ↑	0.72		0.46	0.39	0.35 – 0.93
Random Glucose (mg/dL)	184 ↑	117	101			70 – 140
Sodium (mmol/L)	140.3	142.1	142.6	140.9		135 – 147
Potassium (mmol/L)	3.90	3.14 ↓	3.92	3.36 ↓		3.5 – 5
Chloride (mmol/L)	100.7	102.6	98.5	100.2		94 – 111
Albumin (g/dL)			3.20		2.4	3-5.2
C-Reactive Protein (mg/dL)			42.0			0-6
HBsAg Rapid				Non reactive		
Anti HIV Rapid				Non reactive		

Table 3. Arterial blood gas

Parameter	Feb 18 th	Feb 21 st	Feb 22 nd	Feb 23 rd	Feb 24 th	Feb 25 th
pH	7.38	7.50	7.36	7.49	7.53	7.35
pCO ₂ (mmHg)	47	44	64.7	42.2	39.4	54.2
pO ₂ (mmHg)	72	84	38	119	127	103
HCO ₃ ⁻ (mmol/L)	27.4	34.4	36.1	31.5	32.6	29.1
BE (mmol/L)	1.9	10.3	10.7	7.5	9.3	3.5
TCO ₂ (mmol/L)	29	36	38	33	34	31
O ₂ Saturation (%)	94	97	68	99	99	97

On the same day, the patient experienced clinical deterioration marked by decreased consciousness and increased respiratory distress. Lung examination revealed crackles in both lung fields. Arterial blood gas

(ABG) analysis showed hypoxemia consistent with impending type 1 respiratory failure, prompting transfer to the intensive care unit (ICU). The patient received high-flow oxygen via a non-rebreathing mask (NRM) at 15 L/min, chest physiotherapy, and continued supportive care. Antibiotic therapy was escalated to intravenous meropenem 1 g every 8 hours administered as a 3-hour infusion, resulting in initial clinical improvement.

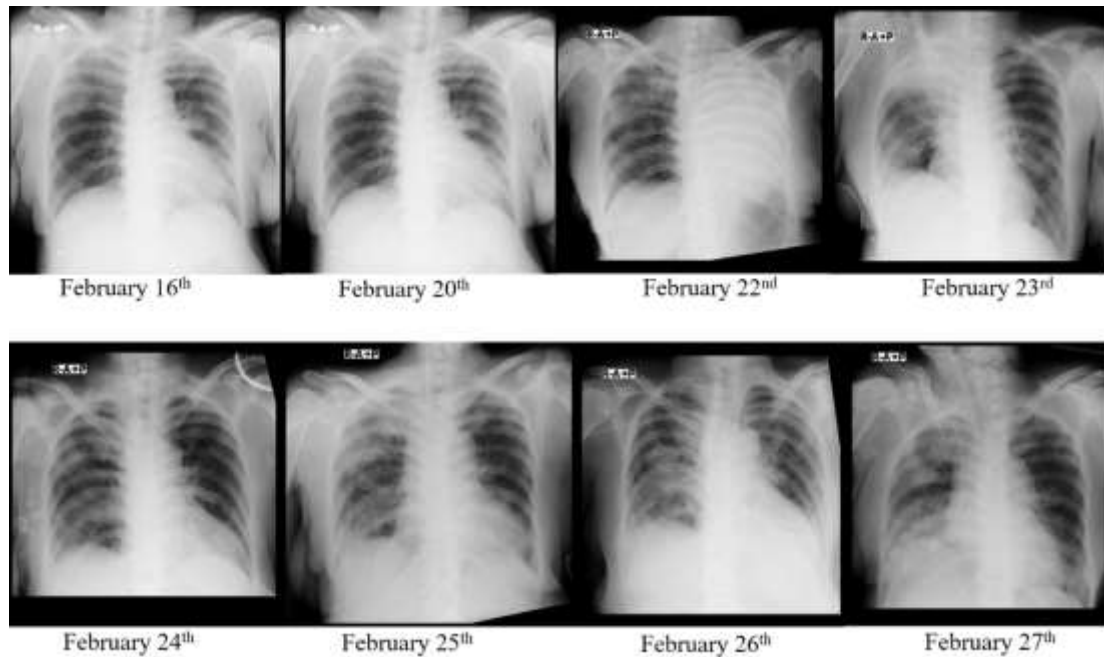


Figure 1. Serial chest x-ray

However, on the sixth day of hospitalization, the patient developed sudden deterioration characterized by severe desaturation, increased work of breathing, and decreased level of consciousness. ABG analysis revealed mixed respiratory failure with severe hypoxemia. Post-intubation chest radiography demonstrated opacification of the left hemithorax with ipsilateral tracheal deviation, suggestive of atelectasis. The patient was diagnosed with severe pneumonia (PSI class V without risk factors for multidrug-resistant organisms), mixed-type respiratory failure, moderate acute respiratory distress syndrome (ARDS), sepsis, and atelectasis secondary to a mucus plug.

Further management included endotracheal intubation and mechanical ventilation, vasopressor support, sedation, and comprehensive supportive interventions, including lung recruitment maneuvers, intensive chest physiotherapy, regular suctioning, oral hygiene, implementation of a ventilator-associated pneumonia (VAP) bundle, and positioning strategies (right lateral decubitus and supine with head elevation of 30°).

Subsequently, the patient's condition gradually improved, as evidenced by better oxygenation and hemodynamic stability. Sputum culture revealed *Enterobacter cloacae* sensitive to meropenem. The patient was successfully weaned from mechanical ventilation and extubated on the eighth day of hospitalization, although tachypnea and ineffective cough persisted.

Due to ongoing impaired airway clearance and secretion retention, a tracheostomy was performed. Following the procedure, the patient showed progressive clinical improvement, including reduced sputum production, improved respiratory pattern, and stable hemodynamic status and body temperature. By the

tenth day of hospitalization, the patient was conscious, responsive to commands, with adequate oxygenation via tracheostomy and controlled signs of infection.

Case Discussion

This case illustrates a complex and multifactorial clinical course in a patient with neurological impairment who developed severe pneumonia complicated by respiratory failure, ARDS, sepsis, and mucus plug-induced atelectasis. In elderly populations, pneumonia carries a high burden not only in terms of acute morbidity and mortality but also long-term outcomes. Previous studies have shown that mortality remains high even after hospital discharge, with up to 54.9% of patients dying within one year, and frailty as well as neurological comorbidities being major contributors to poor prognosis.² Furthermore, patients with aspiration pneumonia tend to present with more severe neurological deficits and experience longer hospital stays compared to those without aspiration.³

In this case, the patient's history of ischemic stroke, vascular dementia, and prolonged immobilization significantly contributed to impaired airway protection and clearance. Neurological deficits such as aphasia, quadriparesis, and cognitive impairment are well-known risk factors for dysphagia and ineffective cough, both of which predispose patients to aspiration pneumonia. Aspiration pneumonia occurs when swallowing dysfunction allows oropharyngeal or gastric contents to enter the lower respiratory tract, often in the form of silent aspiration without overt coughing or choking, especially in patients with central nervous system disorders.⁴ Dysphagia is highly prevalent in severe stroke and has been consistently associated with increased risk of pneumonia, prolonged hospitalization, and higher mortality.³

In addition, elderly patients frequently exhibit atypical or subtle clinical manifestations of pneumonia, such as decreased appetite, fatigue, or mild fever, which may delay diagnosis and treatment.⁴ Communication impairment further complicates clinical recognition, as patients may be unable to report symptoms such as swallowing difficulty or dyspnea.⁵ Moreover, prolonged bedridden status contributes to reduced mucociliary clearance, respiratory muscle deconditioning, and secretion retention, all of which exacerbate the risk of pulmonary infection. These factors collectively explain the progression from initial pneumonia to severe respiratory complications in this patient.

The clinical course in this case progressed to respiratory failure, ARDS, and sepsis, reflecting a severe systemic inflammatory response. Sepsis is characterized by a dysregulated host response to infection, resulting in widespread release of inflammatory mediators and endothelial injury. In sepsis-induced ARDS, inflammatory cascades lead to damage of pulmonary capillary endothelium, increased vascular permeability, and activation of immune cells within the lung parenchyma, ultimately resulting in diffuse alveolar damage and impaired gas exchange.⁶ Pneumonia is a major precipitating factor of ARDS, and pneumonia-associated ARDS has been associated with worse oxygenation, longer duration of mechanical ventilation, and higher mortality rates compared to other etiologies.⁷

A critical complication in this patient was atelectasis caused by mucus plugging, which played a central role in the deterioration of respiratory function. Atelectasis is defined as a loss of lung volume and is radiologically characterized by increased opacity, bronchovascular crowding, mediastinal shift toward the affected side, rib space narrowing, and compensatory hyperinflation of the contralateral lung.⁸ In this case, the presence of mucus plug obstructing the airway led to distal air resorption and collapse of lung segments, thereby worsening hypoxemia through increased intrapulmonary shunting.

The pathophysiology of mucus plugging in this patient is closely associated with impaired airway secretion clearance. Cough plays a critical role in the expectoration of sputum; however, its effectiveness

is often diminished in older adults, particularly in those with neurological disorders. In such conditions, impaired cough mechanics and reduced cough sensitivity are frequently accompanied by dysphagia, both of which significantly increase the risk of aspiration pneumonia. The coexistence of these impairments facilitates aspiration of pathogen-containing saliva and food particles due to increased penetration at the level of the vocal cords. Furthermore, ineffective cough limits the clearance of these materials, thereby prolonging exposure of the airway mucosa to microbial colonization.⁹ In mechanically ventilated patients, physiological airway clearance mechanisms, including mucociliary transport and effective coughing, are further compromised by the presence of artificial airways, insufficient humidification, and patient immobility. Consequently, secretions tend to accumulate within the bronchial tree, establishing a vicious cycle characterized by airway obstruction, ventilation–perfusion mismatch, increased work of breathing, and progressive secretion retention. Although endotracheal suctioning is routinely employed, its effect is largely limited to the proximal airways and does not adequately address distal bronchial secretions, thereby underscoring the need for adjunctive airway clearance strategies.¹⁰

Flexible bronchoscopy is considered the gold standard for the management of mucus plug–induced atelectasis, particularly in cases of persistent lobar collapse or severe hypoxemia.⁸ However, in resource-limited settings where bronchoscopy is not available, alternative strategies must be employed. In this context, chest physiotherapy plays a crucial role as a non-invasive and effective intervention for secretion clearance and lung re-expansion. Evidence suggests that the effectiveness of chest physiotherapy, including postural drainage, percussion, vibration, and lung expansion techniques, may be comparable to bronchoscopy in selected cases.⁸

Chest physiotherapy is part of a broader group of airway clearance techniques (ACTs), which aim to reduce airway obstruction, mobilize secretions from peripheral to central airways, and facilitate expectoration or suctioning.¹¹ These techniques improve ventilation, enhance gas exchange, and reduce the risk of infection by preventing secretion accumulation. Physiologically, percussion and vibration generate mechanical energy that dislodges mucus from the bronchial walls, while postural drainage utilizes gravity to facilitate the movement of secretions from distal lung segments toward the central airways.¹²

In addition to conventional techniques, structured methods such as the Active Cycle of Breathing Techniques (ACBT) have been shown to enhance secretion clearance. ACBT combines breathing control, thoracic expansion exercises, and forced expiratory techniques to improve airflow behind retained secretions, promote collateral ventilation, and facilitate mucus mobilization while minimizing airway collapse.¹¹ Other adjunctive strategies, including nebulization, oscillatory devices, and early mobilization, further support airway clearance and have been associated with improved pulmonary function, reduced dyspnea, and shorter hospital stays in pneumonia patients.¹³ In severe cases of atelectasis, frequent application of chest physiotherapy, even every two hours, is recommended to optimize outcomes.⁸

Importantly, prior evidence supports the effectiveness of chest physiotherapy in resolving mucus plug–related atelectasis. A case reported by Kaur et al. (2020) demonstrated significant clinical and radiological improvement following bedside chest percussion in a patient with lobar atelectasis due to mucus plugging, without the need for bronchoscopy. This finding is consistent with the clinical course observed in this patient, where intensive chest physiotherapy, combined with suctioning and optimal positioning, resulted in gradual improvement in oxygenation, reduction in sputum burden, and re-expansion of the affected lung.¹⁴

Beyond its role in acute management, chest physiotherapy also contributes to improved respiratory muscle function, enhanced cough effectiveness, and overall functional recovery. It has been shown to improve oxygen saturation, reduce dyspnea, and enhance quality of life in patients with pneumonia.¹³ This is particularly relevant in patients with neurological impairment, where restoration of airway clearance mechanisms is essential to prevent recurrent pulmonary complications.

Overall, this case underscores the critical importance of early recognition of secretion retention and aggressive airway clearance management in patients with neurological disorders and pneumonia. In settings where bronchoscopy is not feasible, chest physiotherapy serves as a vital, evidence-based alternative for the management of mucus plug-induced atelectasis. The integration of multidisciplinary care, including respiratory support, infection control, and rehabilitation strategies, is essential to optimize outcomes in this vulnerable population.

Conclusion:

Chest physiotherapy plays a crucial role in the management of mucus plug-induced atelectasis, particularly in patients with neurological impairment and ineffective airway clearance. In this case, intensive airway clearance strategies contributed to clinical and radiological improvement, despite the absence of bronchoscopy. Early recognition of secretion retention and timely intervention are essential to prevent further respiratory complications and improve patient outcomes.

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