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Fatal Severe Community-Acquired Pneumonia with Sputum Retention in a 19-Year-Old Woman with Post-Poliomyelitis Syndrome: A Case Report

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A B S T R A C T

Introduction: Post-poliomyelitis syndrome (PPS) is a late complication of paralytic poliomyelitis in which progressive failure of enlarged motor units extends to the respiratory muscles, eroding ventilatory reserve and the capacity to clear airway secretions. Fatal pneumonia in a young adult with PPS is rarely documented; we describe such a case to highlight the role of diminished respiratory reserve and sputum retention.

Case presentation: A 19-year-old Indonesian woman with childhood paralytic poliomyelitis, generalized atrophy, thoracolumbar scoliosis, and pectus carinatum presented with one hour of acute dyspnoea preceded by three days of productive cough and fever. She was febrile (38.8 C), tachypnoeic, and hypoxaemic (PaO_2 58 mmHg; SpO_2 93% on room air), with bilateral crackles, severe leukocytosis (30,100/microL), mild anaemia (haemoglobin 9.0 g/dL), and Gram-positive cocci on sputum microscopy. Despite empirical antibiotics, bronchodilators, mucolytics, fluids, and oxygen, she deteriorated within 24 hours, requiring intubation and mechanical ventilation; repeat blood gas showed a profound metabolic acidosis (pH 6.914, base excess -26.5 mmol/L). She developed vasopressor-dependent septic shock with multi-organ failure and died on the sixth hospital day.

Conclusion: The diminished respiratory reserve, ineffective cough, and impaired airway clearance intrinsic to PPS can convert an otherwise moderate community-acquired pneumonia into rapidly fatal respiratory failure and septic shock; such pneumonia warrants early severity stratification, aggressive airway clearance, and a low threshold for ventilatory support.

1. Introduction

Poliomyelitis is an acute viral infection caused by poliovirus, an enterovirus transmitted predominantly by the faecal-oral route. After replicating in the gastrointestinal tract, the virus may invade the central nervous system and destroy lower motor neurons, particularly the anterior horn cells of the spinal cord, producing the asymmetric acute flaccid paralysis that defines the paralytic form of the disease.[1] The loss of these motor neurons underlies the muscle weakness, fatigue, and pain that may persist for decades and, in a large proportion of survivors, re-emerge later in life as post-poliomyelitis syndrome (PPS).[1]

Following the introduction of the inactivated poliovirus vaccine by Salk in 1955 and the oral vaccine by Sabin in 1962, the incidence of

poliomyelitis fell dramatically, and the Global Polio Eradication Initiative has since reduced the worldwide burden of wild poliovirus by more than 99%.[2] Nevertheless, an estimated tens of millions of polio survivors remain alive, and many begin to report new neuromuscular decline years to decades after an apparently stable convalescence.[1] This phenomenon, first widely recognized in the late 1970s and 1980s among survivors infected during the mid-century epidemics, is now understood as PPS: a clinical diagnosis requiring a documented history of paralytic poliomyelitis, partial or complete neurological recovery followed by a prolonged period of stability, and the subsequent onset of new muscle weakness or abnormal fatigability not explained by other conditions.[1,3] In regions where vaccination coverage has historically been incomplete, including parts of Indonesia, a reservoir of polio survivors persists, and vigilance for acute flaccid paralysis remains a cornerstone of global surveillance.[2]

In Indonesia, as in several other low- and middle-income settings, historical gaps in immunization coverage left successive birth cohorts incompletely protected against wild poliovirus, and the country has continued to confront sporadic outbreaks linked to under-vaccinated communities. The survivors of these infections now constitute an aging population at risk of late neuromuscular deterioration, frequently without the benefit of structured long-term follow-up. Many, like the patient described here, never received formal rehabilitation and carry fixed deformities and respiratory vulnerabilities that remain clinically silent until an acute stressor intervenes. Recognition of this latent burden is important for internists and pulmonologists, who may encounter polio survivors presenting acutely many years after the original diagnosis and in whom the respiratory consequences of the disease are easily underestimated.[3]

The pathophysiology of PPS is complex and centres on chronic denervation and reinervation. After the acute insult, surviving motor neurons compensate by collateral sprouting, generating abnormally enlarged motor units that maintain muscle function for many years. Over time, however, these overextended units incur a sustained metabolic burden and begin to fail, producing further denervation, muscular atrophy, and progressive loss of strength; physiological aging and low-grade inflammation accelerate this decline.[1,3] Crucially, the lower motor neuron injury of poliomyelitis is not confined to the limbs. The motor units serving the diaphragm, intercostal muscles, and the expiratory musculature may also be depleted, so that respiratory muscle strength and endurance decline even when conventional spirometric indices remain within the normal range.[3,4]

This subclinical respiratory compromise has two consequences of particular relevance to acute illness. First, the reduced ventilatory reserve becomes manifest only under stress, such as a lower respiratory tract infection, when the work of breathing rises and the weakened respiratory pump cannot keep pace, predisposing to ventilatory failure.[1] Second, weakness of the expiratory muscles undermines the cough, the principal mechanism for clearing bronchopulmonary secretions. An ineffective cough leads to retention of secretions in the distal airways, atelectasis, bacterial colonization, and a heightened risk of pneumonia, a sequence well documented across the spectrum of neuromuscular disease.[5,6] Coexisting thoracic cage deformity, such as scoliosis and pectus carinatum, further restricts lung expansion and compounds the restrictive ventilatory defect.[7] Bulbar involvement, when present, adds the risk of recurrent micro-aspiration. Together these mechanisms make pneumonia in the polio survivor a potentially catastrophic event rather than the self-limiting illness it often represents in the general population.[4]

The spectrum of respiratory involvement in PPS ranges from clinically inapparent reductions in respiratory muscle endurance to overt chronic hypoventilation requiring assisted ventilation. Sleep-disordered breathing, nocturnal hypoventilation, and a blunted capacity to augment minute ventilation are recognized features that tend

to progress insidiously. Because resting gas exchange is frequently normal, the magnitude of the underlying deficit is easily underestimated until a febrile illness, with its attendant rise in metabolic and ventilatory demand, exposes the narrow reserve. This pattern of compensated physiology that fails abruptly under load is a recurring theme in neuromuscular respiratory medicine and frames the interpretation of the present case.[6,7]

Community-acquired pneumonia (CAP) remains a leading infectious cause of death worldwide, and Streptococcus pneumoniae is the single most frequently identified pathogen in adults requiring hospitalization.[8,9] Yet the literature describing severe pneumonia with a fatal outcome in young adults with PPS is remarkably sparse, and the condition is seldom considered in routine risk stratification. Most published reports on PPS address rehabilitation, chronic ventilatory support, or perioperative management rather than the acute trajectory of an intercurrent pulmonary infection.[1,3]

The novelty of the present report lies in its detailed documentation of the rapid, stepwise decompensation from community-acquired pneumonia to refractory septic shock and death in a 19-year-old woman with PPS and severe chest-wall deformity, a confluence of host vulnerabilities rarely captured in a single case. The aim of this report is to describe the clinical course and to highlight the mechanistic role of diminished respiratory reserve, ineffective cough, and sputum retention as drivers of fatal respiratory failure in PPS, so that clinicians may recognize and act upon this high-risk phenotype earlier.

2. Case Presentation

History and presenting complaint

A 19-year-old Indonesian woman with a history of poliomyelitis presented to the emergency department with acute shortness of breath that had begun approximately one hour before arrival. The dyspnoea was preceded by three days of productive cough and one day of intermittent fever. She reported no chest pain, haemoptysis, nausea, or vomiting. Her baseline demographic and clinical characteristics are summarized in Table 1.

Her medical history was notable for weakness of all four limbs dating from the age of one year. At eight years of age she received a diagnosis of poliomyelitis. After the acute illness she underwent no formal medical rehabilitation and had not completed her basic childhood immunization schedule. She had been unable to walk since childhood and was severely limited in physical activity. Over the course of her disease she developed generalized muscular atrophy, contractures of the lower limbs, and musculoskeletal deformity comprising thoracolumbar scoliosis and pectus carinatum. There was no history of other chronic illness, no regular medication use, and no known drug allergy.

Physical examination findings

On examination the patient appeared moderately to severely ill; her vital signs and the salient physical findings are included in Table 1. She was febrile at 38.8 degrees C, with a heart rate of 115 beats per minute and a respiratory rate of 29 breaths per minute. Peripheral oxygen saturation was 93% on room air and rose to 100% with supplemental oxygen delivered by nasal cannula. Blood pressure was within normal limits. Auscultation of the chest revealed bilateral coarse crackles. Neurological examination demonstrated generalized muscular atrophy, severe motor weakness of all four limbs, areflexia, and joint contractures; the cranial nerves were intact.

Laboratory and arterial blood gas findings

Initial laboratory and arterial blood gas investigations are presented with reference ranges in Table 2. They revealed severe leukocytosis with a white blood cell count of 30,100/microL, reflecting an intense immune response to acute infection, accompanied by mild anaemia with a haemoglobin of 9.0 g/dL attributable to chronic inflammation or secondary malnutrition in the setting of long-standing neuromuscular disease. Renal function testing showed an elevated urea of 36.2 mg/dL without a significant rise in creatinine, a pattern

consistent with prerenal acute kidney injury secondary to hypoperfusion. Microscopic examination of the sputum identified Gram-positive cocci at 10-15 per low-power field, supporting a bacterial pneumonia as the principal infective process.

Table 1. Clinical characteristics of the patient.

Characteristic	Value
Age	19 years
Gender	Female
Ethnicity / nationality	Indonesian
Relevant history	Paralytic poliomyelitis diagnosed at age 8; limb weakness from age 1
Functional status	Non-ambulant since childhood; severely limited physical activity
Rehabilitation / immunization	No formal rehabilitation; incomplete basic immunization
Chronic deformity	Generalized muscular atrophy, lower-limb contractures, thoracolumbar scoliosis, pectus carinatum
Comorbidity / medication / allergy	None known
Chief complaint	Acute dyspnoea (1 hour) with 3 days of productive cough and 1 day of fever
Temperature	38.8 C* (normal 36.5-37.5 C)
Heart rate	115 beats/min* (normal 60-100)
Respiratory rate	29 breaths/min* (normal 12-20)
Blood pressure	Within normal limits
Oxygen saturation (room air)	93%* (rose to 100% on nasal cannula)
Chest auscultation	Bilateral coarse crackles*
Neurological	Generalized atrophy, severe quadriparesis, areflexia, contractures; cranial nerves intact*

*Notes: Values marked * lie outside the normal reference range or denote an abnormal finding.*

Table 2. Laboratory and arterial blood gas findings on admission and during intensive care, with reference ranges.

Parameter	Result	Reference range
White blood cell count	30,100/microL*	4,000-11,000/microL
Haemoglobin	9.0 g/dL*	12.0-15.0 g/dL
Urea	36.2 mg/dL*	15-40 mg/dL
Creatinine	Not significantly elevated	0.6-1.1 mg/dL
Sputum microscopy	Gram-positive cocci, 10-15/LPF*	No pathogenic organisms
ABG PaO_2 (room air, FiO_2 0.21)	58 mmHg*	80-100 mmHg
ABG PaO_2 (after nasal cannula)	95 mmHg	80-100 mmHg
ABG pH (intensive care)	6.914*	7.35-7.45
ABG $PaCO_2$ (intensive care)	26.8 mmHg*	35-45 mmHg
ABG base excess (intensive care)	-26.5 mmol/L*	-2 to +2 mmol/L
Oxygen saturation (room air)	93%*	>=95%

*Notes: LPF, low-power field; ABG, arterial blood gas; FiO_2, fraction of inspired oxygen; PaO2, arterial oxygen tension; $PaCO_2$, arterial carbon dioxide tension. Values marked * lie outside the reference range. The dissociation between an elevated urea and a preserved creatinine supports prerenal azotaemia from hypoperfusion.*

Arterial blood gas analysis was performed serially and is summarized in Table 2 and illustrated in Figure 1. As shown in Figure 1A, the initial sample, drawn on room air (FiO_2 21%), demonstrated hypoxaemia with a PaO_2 of 58 mmHg and an oxygen saturation of 93%, improving

markedly to a PaO_2 of 95 mmHg after the institution of nasal-cannula oxygen. On the first hospital day the patient deteriorated and required endotracheal intubation and mechanical ventilation. A repeat arterial blood gas during intensive care revealed a profound metabolic acidosis, detailed in Figure 1B, with a pH of 6.914 and a $PaCO_2$ of 26.8 mmHg, the latter reflecting maximal respiratory compensation through hyperventilation, together with a base excess of -26.5 mmol/L (Figure 1C). This biochemical picture is characteristic of a lactic acidosis arising from septic shock and tissue hypoperfusion. Taken together, the laboratory and blood-gas profile depicted a severe physiological insult imposed by an acute respiratory infection upon a markedly limited ventilatory reserve.

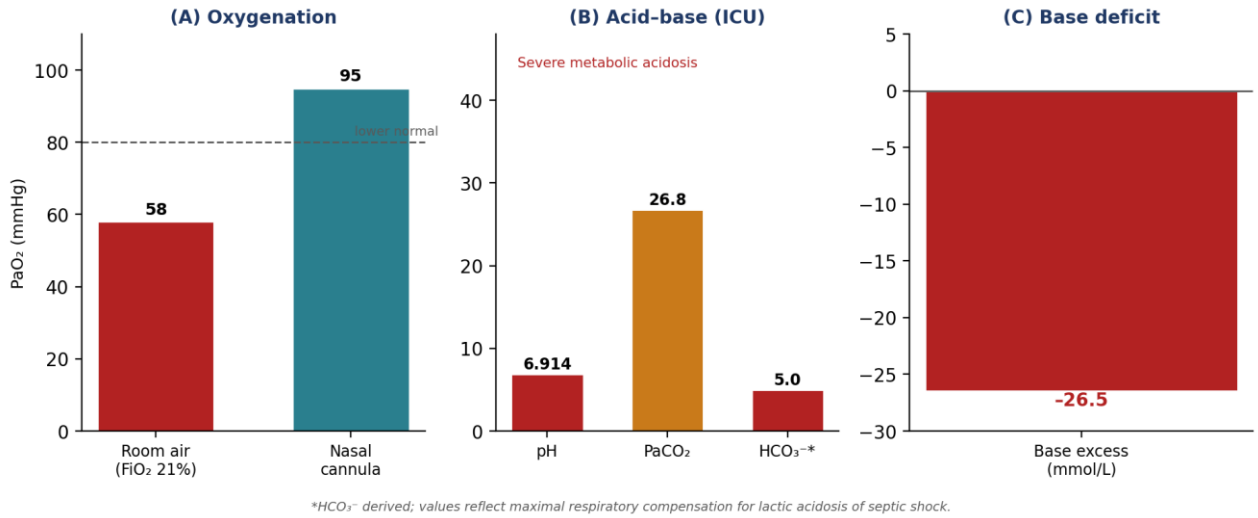

Figure 1. Arterial blood gas trajectory. (A) Oxygenation improved from a PaO2 of 58 mmHg on room air to 95 mmHg with nasal-cannula oxygen. (B) During intensive care the patient developed a severe metabolic acidosis (pH 6.914) with a compensatory reduction in $PaCO_2$. (C) The base deficit reached -26.5 mmol/L, consistent with the lactic acidosis of septic shock.

Diagnosis, management, and outcome

The clinical reasoning at admission centred on an acute lower respiratory tract infection superimposed on chronic neuromuscular disease. The combination of fever, productive cough, bilateral crackles, marked leukocytosis, hypoxaemia, and Gram-positive cocci on sputum microscopy pointed firmly to a bacterial pneumonia, most probably pneumococcal. Alternative contributors were weighed: aspiration related to possible bulbar dysfunction, given the neuromuscular background; and a primary ventilatory failure from respiratory muscle fatigue, suggested by the thoracic deformity and the rapidity of the deterioration. Cardiac failure was considered unlikely in view of the normal blood pressure and the absence of a relevant cardiac history, and the preserved creatinine argued against established intrinsic renal disease. The working diagnosis therefore remained severe bacterial pneumonia precipitating acute-on-chronic respiratory failure, with the neuromuscular substrate amplifying each step of the decline.

The patient was diagnosed with acute pneumonia upon a background of post-poliomyelitis syndrome and was treated with empirical intravenous antibiotics, a bronchodilator, a mucolytic, intravenous fluids, and supplemental oxygen. Within the first 24 hours of admission her condition worsened, with increasing respiratory distress and a declining level of consciousness, prompting endotracheal intubation and mechanical ventilation. On the third hospital day she developed haemodynamic instability requiring vasopressor support. Her condition then progressed to septic shock with features of multi-organ failure, manifested by impaired peripheral perfusion, a depressed level of consciousness, and an escalating

requirement for inspired oxygen during mechanical ventilation. She died on the sixth hospital day owing to progressive clinical deterioration. The complete clinical course and management are summarized in Table 3.

Table 3. Clinical course and management timeline.

Hospital day	Clinical events and management
Day 0 (admission)	Acute dyspnoea, fever 38.8 C, respiratory rate 29/min, SpO_2 93% on room air; white cell count 30,100/microL; PaO_2 58 mmHg. Started on empirical intravenous antibiotics, a bronchodilator, a mucolytic, intravenous fluids, and supplemental oxygen.
Day 1	Clinical deterioration with increasing respiratory distress and declining consciousness; endotracheal intubation and mechanical ventilation.
Day 3	Haemodynamic instability requiring vasopressors; vasopressor-dependent septic shock with multi-organ failure; arterial pH 6.914, base excess -26.5 mmol/L.
Day 6	Progressive clinical deterioration and multi-organ failure; death.

Notes: SpO_2, peripheral oxygen saturation; PaO2, arterial oxygen tension.

In summary, this case demonstrates that a patient with PPS and severe thoracic deformity possesses a critically low respiratory reserve and is therefore prone to rapid decompensation when an acute lower respiratory tract infection supervenes. A pneumonia that might be managed uneventfully in a healthy individual progressed in this young woman to respiratory failure and septic shock, underscoring the importance of early recognition, aggressive respiratory support, and close monitoring in this high-risk group.

3. Discussion

Post-poliomyelitis syndrome is a lower motor neuron disorder that arises from chronic denervation and reinervation after the acute poliovirus infection. Destruction of motor neurons in the anterior horn of the spinal cord causes the loss of functional motor units; the surviving neurons compensate through collateral sprouting, forming abnormally enlarged motor units. Over time these metabolically overburdened units fail, producing further denervation, atrophy, and a progressive decline in muscle strength.[1] PPS is increasingly understood as a multi-system disorder rather than a purely motor one, with contemporary studies documenting accompanying somatosensory dysfunction and chronic pain in affected survivors, underscoring the diffuse and progressive nature of the late polio phenotype.[10] The diagnosis of the antecedent poliomyelitis is essentially clinical, resting on a picture of acute, flaccid, asymmetric paralysis without sensory loss, classically with a predilection for the lower limbs and often accompanied by fever in the prodrome.[2] Because only a small minority of poliovirus infections progress to the paralytic form, every case of acute flaccid paralysis is treated within global surveillance systems as poliomyelitis until proven otherwise, a principle that has underpinned the eradication effort.[2] Our patient's lifelong non-ambulant quadriparesis, areflexia, and generalized atrophy are entirely consistent with severe paralytic poliomyelitis acquired in early childhood and its subsequent progression.

At the cellular level, the long-term fate of the reinnervated motor unit explains the progressive nature of PPS. After poliovirus destroys a large fraction of anterior horn cells, the few surviving motor neurons sprout new axonal branches to reinnervate orphaned muscle fibres, sometimes enlarging a single motor unit several-fold. This compensation restores function but imposes a

chronic metabolic load on the parent neuron and its terminal arborization. Decades later, the most distal sprouts begin to fail, producing a slow, ongoing process of denervation that outpaces continued reinnervation; the clinical correlate is new weakness and fatigability in muscles once thought to have recovered. When this process involves the motor units of the respiratory muscles, the ceiling of ventilatory performance falls, even though the loss may remain invisible on routine spirometry until reserve is tested by acute illness.[1,3]

The lower motor neuron injury of PPS extends beyond the limbs to the muscles of respiration, including the diaphragm, the intercostals, and the expiratory muscles. Depletion of the motor units innervating these muscles reduces respiratory strength and endurance, a deficit that is frequently subclinical at rest. Under the stress of an acute lower respiratory tract infection, however, the limited ventilatory reserve becomes clinically apparent and can progress rapidly to ventilatory failure.[3-5] In neuromuscular disease, including post-polio syndrome, resting pulmonary function and static spirometric indices may be relatively preserved, yet the maximal cardiorespiratory and exercise capacity is measurably reduced, reflecting a constrained ventilatory and aerobic reserve and reduced endurance of the respiratory muscles rather than a fixed structural defect.[3] This dissociation, whereby maximal performance is limited while resting function appears normal, is precisely the substrate that predisposes to impaired airway clearance and to the amplification of an intercurrent pneumonia under acute stress.[4]

Weakness of the expiratory muscles is central to the impairment of cough in PPS. An ineffective cough cannot generate the expiratory flow required to shear secretions from the airway wall, so that bronchopulmonary secretions accumulate in the distal airways. This persistent sputum retention impairs alveolar ventilation, promotes atelectasis, and facilitates colonization by pathogenic organisms, thereby raising the risk of severe pneumonia.[5] The relationship between cough strength and the burden of respiratory infection is well established across neuromuscular disease, and objective measures of cough efficacy, such as peak cough flow, are used to time the introduction of airway-clearance and cough-augmentation techniques such as mechanical insufflation-exsufflation.[5,11] Beyond the cough, the mucociliary escalator provides a continuous baseline mechanism for clearing inhaled particles and microorganisms; when an effective cough can no longer supplement this baseline clearance, the lung becomes increasingly vulnerable to infection. Coexisting bulbar weakness, with the attendant risk of recurrent micro-aspiration, compounds this vulnerability and is itself associated with aspiration pneumonia in the critically ill.[6] These mechanisms explain why lower respiratory tract infections in PPS frequently follow a more severe course than in individuals without neuromuscular disease.

The patient's restrictive physiology was further aggravated by her thoracic cage deformity. Scoliosis and pectus carinatum reduce the compliance of the chest wall and limit lung expansion, adding a structural restrictive defect to the neuromuscular one. In patients with neuromuscular disease and rib-cage deformity, this combination predisposes to nocturnal hypoventilation and, ultimately, to chronic hypercapnic respiratory failure; the progressive decline in respiratory function in such patients is an accepted indication for home non-invasive ventilation and warrants structured respiratory surveillance.[7] Although our patient had not undergone such surveillance, her deformity almost certainly narrowed the physiological margin available to withstand an acute infective insult, helping to explain the speed of her decompensation. As shown in Figure 2, these vulnerabilities act in series: the neuromuscular and structural defects converge to impair cough and clearance, which in turn permit sputum retention and infection, culminating in respiratory failure and shock.

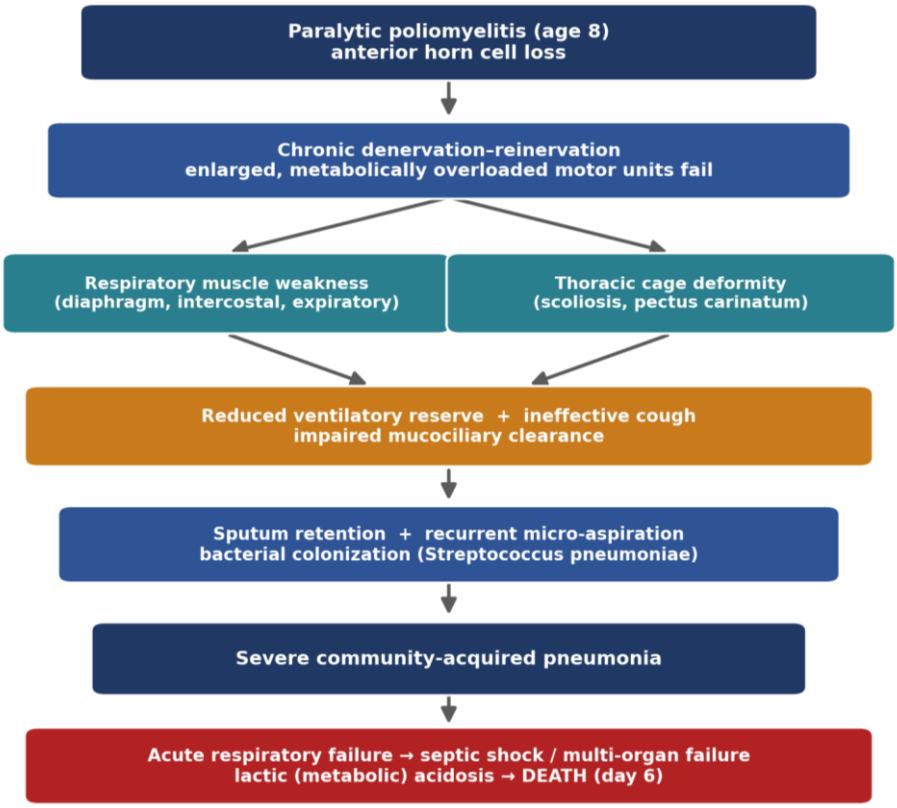

Figure 2. Proposed pathophysiological cascade linking post-poliomyelitis syndrome to fatal pneumonia. Chronic lower motor neuron loss produces respiratory muscle weakness, which together with thoracic cage deformity reduces ventilatory reserve and cough efficacy. The resulting sputum retention and micro-aspiration favour pneumococcal pneumonia, which precipitates respiratory failure, septic shock, and death.

Once pneumonia is established, severity assessment guides the intensity of care. According to the 2019 Infectious Diseases Society of America and American Thoracic Society (IDSA/ATS) guideline, a diagnosis of severe community-acquired pneumonia requires at least one major criterion or three or more minor criteria.[8] The major criteria are respiratory failure requiring invasive mechanical ventilation and septic shock requiring vasopressor support. The minor criteria include a respiratory rate of 30 breaths per minute or more, a PaO_2/FiO_2 ratio of 250 or less, multilobar infiltrates, confusion or disorientation, uraemia (blood urea nitrogen 20 mg/dL or more), leukopenia from infection, thrombocytopenia, hypothermia, and hypotension requiring aggressive fluid resuscitation. Table 4 maps the patient's findings against these criteria. She fulfilled both major criteria, having required intubation and mechanical ventilation within the first 24 hours and having progressed to vasopressor-dependent septic shock, in addition to displaying minor features, including marked hypoxaemia (PaO_2 58 mmHg on FiO_2 0.21, giving a calculated PaO_2/FiO_2 ratio of approximately 276, close to the qualifying threshold of 250), near-threshold tachypnoea (29 breaths/min), and uraemia (urea 36.2 mg/dL). The fulfilment of the major criteria, independent of the minor ones, placed her unequivocally in the severe category from early in the illness and accounts for the rapid progression to respiratory failure and shock, underscoring the need for intensive monitoring and aggressive management in accordance with the guideline.[8] Contemporary cohort studies of severe community-acquired pneumonia confirm that the very features displayed by this patient, namely the need for vasopressors, an elevated body temperature, and a raised blood urea nitrogen, are independent predictors of in-hospital death.[12,13]

Table 4. Application of the 2019 IDSA/ATS criteria for severe community-acquired pneumonia to the patient.

Criterion	Type	Present in this patient
Invasive mechanical ventilation for respiratory failure	Major	Yes - intubated within 24 hours
Septic shock requiring vasopressors	Major	Yes - vasopressor-dependent by day 3
Respiratory rate >=30 breaths/min	Minor	Near threshold (29 breaths/min)
PaO_2/FiO_2 <=250	Minor	Borderline - PaO_2 58 on FiO_2 0.21 (ratio ~276)
Uraemia (BUN >=20 mg/dL)	Minor	Yes - urea 36.2 mg/dL
Multilobar infiltrates	Minor	Bilateral crackles; not radiographically confirmed
Confusion / disorientation	Minor	Developed declining consciousness

Notes: IDSA/ATS, Infectious Diseases Society of America / American Thoracic Society; BUN, blood urea nitrogen. Fulfilment of one major or three minor criteria defines severe community-acquired pneumonia. The patient met both major criteria.

What renders this case particularly instructive is the patient's youth. Community-acquired pneumonia carries its highest mortality among the elderly and those with cardiorespiratory comorbidity, whereas a 19-year-old without conventional risk factors would ordinarily be expected to recover. In this patient, however, the neuromuscular substrate functioned as a powerful, non-traditional risk factor that compressed the usual timeline of severe pneumonia into a matter of days. The trajectory from a self-presentation with preserved blood pressure to intubation within 24 hours, vasopressor-dependent shock by the third day, and death by the sixth illustrates how rapidly reserve can be exhausted when the respiratory pump is already operating near its ceiling. Established severity tools such as the Pneumonia Severity Index and the CURB-65 score predict mortality well across causes of community-acquired pneumonia, yet they do not capture the neuromuscular vulnerability that dominated this case.[14] This observation argues for regarding PPS, and neuromuscular disease more broadly, as an independent marker of high risk in the assessment of pneumonia, irrespective of age.

Management of severe community-acquired pneumonia follows established principles: prompt initiation of appropriate empirical antibiotics, attentive supportive care, and timely escalation of organ support. Guidelines recommend that empirical therapy for severe disease cover Streptococcus pneumoniae and other likely pathogens, with combination regimens and subsequent de-escalation guided by microbiological results and clinical response; delayed or inappropriate initial therapy is associated with worse outcomes.[8,15] In the patient with PPS, however, antimicrobial therapy alone is insufficient, because the pathophysiology is dominated by a failure of secretion clearance and ventilation. The supportive elements of care, including secretion management, ventilatory support, and haemodynamic resuscitation, assume an importance at least equal to that of the antibiotic. The bronchodilator and mucolytic administered to this patient reflect an appropriate intent to mobilize secretions, although in the context of a profoundly weak cough their efficacy is limited without mechanical assistance.

This limitation underlines the potential role of cough-augmentation and airway-clearance techniques. In neuromuscular disease, assisted coughing, both manual and mechanically assisted, air stacking, and mechanical insufflation-exsufflation can raise peak cough flow above the threshold required for effective secretion clearance, and their early use during acute respiratory exacerbations may reduce morbidity.[5,11] Non-invasive ventilation has an established place in the chronic management of restrictive disease from neuromuscular weakness and chest-wall deformity, and a carefully monitored trial of non-invasive support may be appropriate in selected acute presentations, provided that escalation to invasive

ventilation is not unduly delayed.[7] Whether earlier institution of such measures could have altered the course in this patient cannot be known, but the case highlights their conceptual relevance and the value of anticipatory respiratory care in this population.[6]

The microbiological context is consistent with the most probable pathogen. Streptococcus pneumoniae is the commonest cause of community-acquired pneumonia in adults worldwide and remains the dominant organism in severe disease requiring hospitalization.[8,16] Its predominance reflects an ability to colonize the nasopharynx and translocate to the lower airways, particularly in hosts with impaired pulmonary defence.[9] In a patient with PPS, expiratory muscle weakness and ineffective cough lead to inadequate clearance of bronchopulmonary secretions, facilitating micro-aspiration and the colonization of the distal airways by pathogens such as S. pneumoniae. The finding of Gram-positive cocci on sputum microscopy in this patient is therefore best interpreted not as an incidental result but as the microbiological correlate of an impaired host defence, making pneumococcus the most plausible cause of her pneumonia.[16]

The pneumococcus is not only common but also clinically aggressive. In large hospital-based series, pneumococcal pneumonia is associated with a more severe course than non-pneumococcal disease, with higher rates of intensive care admission and mechanical ventilation and a propensity for bacteraemic spread.[16] In vulnerable hosts, pneumococcal pneumonia has been identified as one of the strongest independent predictors of a severe disease course, carrying an odds ratio for death or intensive care admission an order of magnitude above baseline.[17] In a patient with severely limited respiratory reserve, this combination of a virulent organism and a fragile pulmonary defence predisposes to fulminant pneumonia, bacteraemia, and the systemic complications of severe sepsis. The aggressive course observed in our patient is consistent with this interplay between pneumococcal virulence and the host vulnerability conferred by PPS.

The patient's terminal physiology was that of septic shock. Her intensive-care blood gas revealed a profound metabolic acidosis with a markedly negative base excess and a compensatory reduction in $PaCO_2$, a pattern that signifies a type A lactic acidosis driven by tissue hypoperfusion and dysoxia. In critically ill and septic patients, the magnitude of hyperlactataemia and the failure to clear lactate are among the strongest biochemical predictors of death, and a lactate-to-albumin ratio likewise stratifies in-hospital mortality in pneumonia requiring intensive care.[18,19] Despite escalation to mechanical ventilation and vasopressors, the depth of the acidosis in this patient indicated severe, sustained hypoperfusion and carried an ominous prognosis. The accompanying rise in urea without a proportional rise in creatinine is characteristic of prerenal, sepsis-associated acute kidney injury, which typically arises within the first days of sepsis onset and independently increases the risk of death.[20] These findings together describe a young patient overwhelmed by a severe infection that her physiological reserve could not withstand.

The depth of the metabolic derangement merits comment. A pH of 6.914 with a base excess of -26.5 mmol/L represents one of the most severe acidaemias compatible with life and signifies overwhelming anaerobic metabolism from global hypoperfusion. The low $PaCO_2$ reflects the respiratory system's attempt to compensate by eliminating carbon dioxide, a response that is itself constrained in a patient with weak respiratory muscles and that cannot be sustained once fatigue supervenes. In septic shock, the trajectory of lactate, and by extension of the base deficit, is a key indicator of resuscitation adequacy, and a failure to clear lactate despite appropriate therapy portends a poor outcome.[18] In this patient the persistence of profound acidosis despite mechanical ventilation and vasopressor support indicated that perfusion could not be restored, foreshadowing the fatal multi-organ failure that followed. A direct serum lactate concentration was not available for this patient; the inference of a type A lactic acidosis

therefore rests on the severe base deficit together with the clinical context of vasopressor-dependent shock and tissue hypoperfusion.

Host factors beyond the respiratory system also shaped the outcome. The mild anaemia documented at presentation is most consistent with anaemia of chronic disease or nutritional deficiency in the setting of long-standing disability, and it reflects a reduced physiological buffer against the oxygen-delivery demands of severe sepsis; both low and abnormally high haemoglobin concentrations have been associated with increased in-hospital mortality in critically ill septic patients.[21] Chronic immobility and probable undernutrition may further compromise immune competence and respiratory muscle mass, narrowing the margin still further. The early rise in urea with preserved creatinine signals the onset of sepsis-associated, prerenal kidney injury, an early and prognostically important manifestation of the systemic insult in which microvascular dysfunction and inflammation impair renal perfusion before frank tubular damage ensues.[20] Each of these elements, individually modest, compounded to accelerate the patient's decline.

A neuroanatomical perspective reinforces the mechanistic narrative. In poliomyelitis, the injury centres on the lower motor neurons of the anterior horn that innervate skeletal muscle, including the respiratory muscles. Involvement of the motor pathways supplying the diaphragm through the phrenic nerve (C3-C5), together with the intercostal and accessory muscles of the chest wall, reduces ventilatory strength, limits thoracic expansion, and disturbs respiratory mechanics, with consequences for overall lung function.[3] Persistent and progressive neuromuscular dysfunction, compounded by reduced maximal cardiorespiratory capacity and by the chest-wall deformity, further degrades the efficiency of the respiratory pump and its capacity to compensate for an infective stress; the same vulnerability extends to weaning, since inspiratory muscle weakness is a recognized obstacle to liberation from mechanical ventilation in the critically ill.[3,4] This case affirms that a patient with PPS can decompensate rapidly when a lower respiratory tract infection supervenes, even when initial oxygenation appears relatively stable, and therefore requires close monitoring and an aggressive respiratory strategy from the outset.

From a practical standpoint, several learning points emerge. First, pneumonia in a patient with PPS should be regarded as a potentially severe event and stratified for severity early, using objective criteria such as those of the IDSA/ATS, rather than being judged by the apparently reassuring initial oxygenation.[8] Second, because the central deficit is one of secretion clearance, airway-clearance and cough-augmentation strategies, including assisted coughing and mechanical insufflation-exsufflation, together with attention to secretion mobilization, deserve early consideration as adjuncts to antimicrobial therapy in the neuromuscular patient.[5,11] Third, given the limited ventilatory reserve, the threshold for escalating respiratory support, whether non-invasive or invasive, should be low, and such patients are best managed in a setting equipped for close respiratory and haemodynamic monitoring.[7] Finally, the case is a reminder that the legacy of incompletely controlled poliomyelitis persists in communities with historically imperfect vaccination coverage, and that survivors carry lifelong respiratory vulnerability that merits anticipatory care.[1,2]

Prevention deserves emphasis. Patients with neuromuscular disease and chronic respiratory vulnerability are precisely the group in whom immunization against respiratory pathogens, including pneumococcus and influenza, offers the greatest potential benefit, and in whom proactive respiratory assessment can identify declining function before a crisis. Pneumococcal conjugate and polysaccharide vaccines reduce invasive disease, and vaccinating the wider community contributes a degree of herd protection that benefits vulnerable individuals.[16] For polio survivors specifically, periodic evaluation of respiratory muscle strength, cough efficacy, and nocturnal ventilation would permit the timely introduction of supportive measures. The absence of such

structured follow-up in this patient, who had received neither rehabilitation nor complete immunization, mirrors a wider gap in the long-term care of polio survivors in resource-limited settings.[2]

The scarcity of detailed reports of fatal community-acquired pneumonia in young adults with PPS makes direct comparison difficult, and much of the available respiratory literature in PPS concerns chronic ventilatory management or perioperative risk rather than acute infection.[1,3] The mechanistic chain proposed here, from lower motor neuron loss through respiratory muscle weakness and impaired clearance to severe pneumonia and shock, is nevertheless well supported by evidence drawn from the broader neuromuscular population, in whom respiratory infection and respiratory failure are leading causes of death.[5,6] Viewed in that light, the present case is best understood not as an idiosyncratic event but as a predictable, if rarely documented, consequence of severe neuromuscular respiratory compromise meeting a virulent respiratory pathogen.

Limitations

This report has several limitations. As a single case, its clinical findings and outcome cannot be generalized to the entire population of patients with post-poliomyelitis syndrome. Further respiratory investigations, such as spirometry and electromyography or other more comprehensive studies, were not performed during the acute presentation, so the degree of baseline respiratory muscle impairment could not be quantified. In addition, identification of the causative pathogen was limited to sputum microscopy without confirmatory culture or molecular testing, so the microbiological aetiology could not be established definitively. Radiographic confirmation of the distribution of pulmonary infiltrates was likewise not available for inclusion, and a direct serum lactate concentration and a precisely documented time from presentation to first antibiotic dose were not retrievable from the record. Despite these constraints, the report makes an important contribution to understanding the severity of respiratory complications in PPS and emphasizes the need for heightened clinical vigilance in this high-risk group.

4. Conclusion

This case affirms that patients with post-poliomyelitis syndrome possess a critically limited respiratory reserve owing to chronic neuromuscular impairment, respiratory muscle weakness, and thoracic cage deformity, rendering them highly susceptible to rapid respiratory decompensation when a lower respiratory tract infection occurs. A pneumonia that may follow a mild to moderate course in the general population can, in the patient with PPS, progress to severe pneumonia, acute respiratory failure, and septic shock with a rapid and fatal trajectory. Sputum retention arising from an ineffective cough, together with the possibility of recurrent micro-aspiration, constitutes a key mechanism contributing to the severity of pulmonary infection in this setting. Early recognition of pneumonia, vigilant monitoring of respiratory function, proactive airway-clearance measures, and timely ventilatory support and aggressive management are therefore essential in patients with PPS to avert poor clinical outcomes. As observations drawn from a single patient, the management inferences offered here are hypothesis-generating and should be examined in larger studies of polio survivors before being adopted as firm recommendations.

Declarations

Ethics approval and consent to participate: This report was prepared in accordance with institutional ethical standards and the principles of the Declaration of Helsinki. As a retrospective single-case report containing no identifying information, it did not require formal ethics committee review at the authors' institution.

Consent for publication: The patient died during the admission described. Informed consent for the publication of anonymized clinical information was obtained from the patient's next of kin. No identifying details or images are included in this report.

Availability of data and materials: The clinical data supporting this report are contained within the article. Further details are available from the corresponding author upon reasonable request, subject to patient confidentiality.

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