

## Severe Community-Acquired Pneumonia Complicated by Acute Respiratory Distress Syndrome in a Geriatric Patient and Limitation of Life-Sustaining Treatment

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### ABSTRACT

**Introduction:** Severe community-acquired pneumonia (CAP) frequently progresses to acute respiratory distress syndrome (ARDS), imposing high morbidity and mortality rates in patients in advanced stages of life. The confluence of biological frailty, exposure to environmental toxins, and prior corticosteroid use significantly worsens the respiratory prognosis, necessitating careful consideration of the ethical boundaries of futile organ support. **Objective:** To describe the clinical evolution of a geriatric patient with severe CAP and refractory ARDS, analyzing the utilization of non-invasive monitoring scales and the protocolized transition toward the limitation of life-sustaining treatment (LLST) and terminal palliative sedation. **Case Report:** A 92-year-old male with a history of smoking and chronic biomass smoke exposure was admitted due to severe hypoxemic respiratory failure (SpO<sub>2</sub> 78%) and bilateral pulmonary consolidation syndrome. Advanced oxygen therapy via high-flow nasal cannula (HFNC) was implemented, monitored through the ROX Index. Total parenteral nutrition and empirical broad-spectrum double antibiotic coverage were initiated due to the risk of multidrug-resistant (MDR) pathogens and *Pneumocystis jirovecii* secondary to poorly tapered corticosteroid therapy. Following refractory HFNC failure, neurological collapse (Glasgow Coma Scale 3/15), and the presence of advanced signs of hypoperfusion (such as the Schölein-Henoch/Stenon-Louis ocular sign), an interdisciplinary medical consensus meeting was held with the immediate family. Invasive endotracheal intubation was declined, and a legally binding Advance Directive document was signed to transfer the patient to the general ward under a comfort care protocol. **Conclusions:** The terminal palliative protocol—based on continuous infusions of Midazolam, Morphine, and Dexmedetomidine, combined with strict maintenance fluid restriction (Hartmann's solution at 10 mL/h)—completely alleviated refractory dyspnea and prevented terminal respiratory secretions (death rattles), achieving a dignified end-of-life transition. Timely integration of advance directives and monitoring for clinical futility preserve the dignity of terminal geriatric patients.

**KEYWORDS:** Community-Acquired Pneumonia, Adult Respiratory Distress Syndrome, Frail Elderly, Palliative Care, Clinical Evolution.

### ARTICLE DETAILS

**Published On:**  
**11 July 2026**

**Available on:**  
**<https://ijmscrs.com>**

### I. INTRODUCTION

Severe community-acquired pneumonia (CAP) represents one of the infectious pathologies with the greatest epidemiological and healthcare impact within critical care

units worldwide. The progressive increase in global life expectancy has conditioned a notable demographic shift, rising the admission rates of patients belonging to the extreme-geriatric population. In this age group, the loss of

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homeostatic organo-functional reserve and the phenomenon of immunosenescence severely limit the host's adaptive capacity against acute pathogenic insults. The dysregulated pulmonary inflammatory response frequently triggers a local and systemic inflammatory cascade that is exceptionally difficult to control clinically.<sup>1</sup>

Pathophysiologically, the progression of severe CAP toward acute respiratory distress syndrome (ARDS) is driven by the onset of diffuse alveolar damage (DAD), the cardinal histopathological substrate of this entity. This process drastically alters the permeability of the alveolar-capillary membrane, conditioning a massive proteinaceous exudate that inactivates surfactant and occludes dependent lung zones. The consequent diffuse loss of aerated lung tissue increases the intrapulmonary shunt fraction and elevates dead space ventilation. Clinically, this manifests as refractory hypoxemia associated with a marked decrease in respiratory system compliance, necessitating advanced respiratory support.<sup>2</sup>

The implementation of clinical practice guidelines highlights continuous and non-invasive oxygenation monitoring as a fundamental pillar for the early identification of alveolar collapse. The use of peripheral oxygen saturation via pulse oximetry and its ratio to the fraction of inspired oxygen (SpO<sub>2</sub>/FiO<sub>2</sub>) has been globally validated as an effective surrogate for the traditional arterial partial pressure of oxygen ratio (PaO<sub>2</sub>/FiO<sub>2</sub>). These indices, combined with bedside scoring systems, optimize timely respiratory management. A thorough comprehension of these variables facilitates the delineation of therapeutic failure and prevents deleterious delays in crucial clinical decision-making for critically ill patients.<sup>3</sup>

Among non-invasive monitoring strategies in ARDS, the ROX Index has emerged as a clinical tool with high predictive validity, particularly in patients receiving ventilatory support via high-flow nasal cannula (HFNC). This index evaluates the relationship between oxygenation and respiratory rate, objectively identifying subjects who will fail non-invasive management. Early identification of this failure is crucial, as persistent excessive work of breathing generates patient-self-induced lung injury (P-SILI). In the extreme geriatric population, this phenomenon accelerates alveolar damage and multiple organ dysfunction.<sup>4</sup>

Furthermore, the evaluation of individual risk factors in the elderly patient is indispensable to guide empirical antimicrobial therapeutics and predict mechanical complications. Chronic exposure to airway irritants, such as biomass smoke from wood combustion or prolonged smoking, conditions baseline structural changes and a low-grade chronic inflammatory state that destroys ciliary architecture. On the other hand, prior administration of systemic corticosteroids, especially when abruptly discontinued without a dynamic gradual weaning schedule, induces severe secondary tissue immunosuppression. This

vulnerable state depresses alveolar macrophage function, facilitating colonization by opportunistic or multidrug-resistant pathogens.<sup>5</sup>

Nevertheless, when maximal non-invasive therapies fail in patients with extreme frailty or advanced senectud, the multidisciplinary team must confront the bioethical analysis of therapeutic futility. Medical obstinacy (therapeutic hyper-intervention) through invasive maneuvers such as orotracheal intubation or cardiopulmonary resuscitation in terminal phases of disease generates biotrauma and unnecessary suffering without impacting qualitative survival. International consensus promotes the limitation of life-sustaining treatment (LLST) as a formal bioethical intervention to guarantee human dignity and transition toward intensive palliative care. The proper operation of advance directive instruments legally safeguards these decisions, focusing medical efforts exclusively on continuous sedation and comfort for the dying patient.<sup>6</sup>

## II. CASE REPORT

The patient is a 92-year-old male with a relevant medical history of prolonged exposure to biomass smoke from wood combustion for over three decades (averaging 3 to 5 hours daily) and a remote history of smoking (currently discontinued). His remote medical history was notable for a traumatic brain injury that required a trephination surgery approximately 30 years ago, which evolved favorably without residual functional sequelae. Additionally, the patient had a recent 48-hour hospitalization due to left basal pneumonia, subsequently receiving outpatient management with levofloxacin (500 mg every 24 hours for 7 days), systemic steroids (prednisone 20 mg every 24 hours for 2 weeks), and nebulized budesonide. Notably, oral glucocorticoid therapy was abruptly discontinued without an established tapering or dynamic withdrawal schedule. The current illness began two weeks prior to hospital evaluation, characterized by a productive cough and hyaline expectoration. Despite the outpatient treatment administered, the clinical picture failed to improve, evolving hours before admission with asthenia, adynamia, severe hyporexia, and secondary bedridden status due to a total inability to ambulate. Likewise, he manifested dysnea on moderate exertion that rapidly progressed to rest; accompanying gastrointestinal or urinary symptomatology was denied.

Vital signs recorded upon admission to the clinical triage area revealed a blood pressure of 130/86 mmHg, a heart rate of 85 bpm, a respiratory rate of 32 rpm (frank tachypnea), a temperature of 36.0°C, and a peripheral oxygen saturation of 78% on room air, designating an intermediate urgency classification. Upon initial physical examination, the patient was neurologically intact, conscious, and cooperative, with a Glasgow Coma Scale score of 15/15, without clinical data of generalized low cardiac output or initial requirements for vasopressor amines.

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Regarding his respiratory status, he exhibited a restrictive pattern with significant polypnea, but without thoracoabdominal dissociation or evident nasal flaring. Lung auscultation and thoracic palpation detected a frank decrease in vocal fremitus and basal sub-dulness, clinically integrating a bilateral pulmonary consolidation syndrome predominantly on the left side. Heart sounds remained rhythmic, of good intensity, and without audible murmurs, while the abdomen was soft, depressible, non-tender, and with preserved normoperistalsis. Extremities demonstrated mild edema (+), a distal capillary refill time of 3 seconds, and chronic trophic changes characterized by ochre hyperpigmentation in both legs, with the presence of left pretibial hematic crusts due to chronically established peripheral venous insufficiency. Based on these findings, a presumptive diagnosis of severe community-acquired pneumonia versus viral pneumonia was established, associated with acute type 1 (hypoxemic) respiratory failure, clinically categorized under the spectrum of an evolving septic shock.

Non-invasive ventilatory support was immediately initiated with supplemental oxygen therapy via a simple face mask at a flow rate of 5 to 6 L/min, achieving an oxygen saturation recovery to 95% with an estimated fraction of inspired oxygen (FiO<sub>2</sub>) of 36%. The initial pharmacological regimen included fasting, maintenance crystalloid solution infusion at 60 mL/h, paracetamol 1 g IV every 8 hours, micronized nebulizations with budesonide every 12 hours, and ipratropium bromide/salbutamol every 8 hours, omeprazole 40 mg IV every 24 hours, and enoxaparin 40 mg SQ every 24 hours. As empirical broad-spectrum antimicrobial coverage for the risk of multidrug-resistant pathogens due to prior hospitalization and prolonged steroid use, Cefepime 2 g IV every 8 hours was administered in combination with Trimethoprim/Sulfamethoxazole (TMP/SMX) 160/800 mg IV every 8 hours for targeted coverage of *Pneumocystis jirovecii*. Microbiological cultures, a complete blood count, acute phase reactants, and a multiplex respiratory molecular panel via polymerase chain reaction were requested.

Following transfer to the critical monitoring area, initial laboratory results reported mild anemia, leukocytes of  $10.52 \times 10^3/\mu\text{L}$  with neutrophils of  $9.05 \times 10^3/\mu\text{L}$ , moderate thrombocytopenia of  $69 \times 10^3/\mu\text{L}$ , a CRP of 6.89 mg/dL, and procalcitonin of 0.35 ng/mL. Due to persistent shallow tachypnea, support was optimized with a high-flow nasal cannula (HFNC) at 50 L/min and FiO<sub>2</sub> at 40%, subsequently down-titrated to a FiO<sub>2</sub> of 33% with a saturation of 94% and a favorable ROX Index of 13.5. Acute urinary retention secondary to a bladder globe was diagnosed and resolved via a transurethral Foley catheter, and the FilmArray respiratory molecular panel returned negative results for all standardized microorganisms.

The following day, the patient manifested clinical respiratory deterioration with a severe increase in work of breathing and refractory polypnea. Consequently, HFNC parameters were

titrated to the maximum (flow of 60 L/min and FiO<sub>2</sub> at 60%), maintaining with difficulty a borderline saturation of 91% in the context of rising leukocytosis ( $11.79 \times 10^3/\mu\text{L}$ ). Finally, the patient met the clinical and imaging criteria for severe acute respiratory distress syndrome (ARDS), requiring a critical adjustment of the HFNC to a flow rate of 60 L/min and a FiO<sub>2</sub> of 75% to achieve a saturation of 91% amidst erratic respiratory rates (16–30 rpm). Simultaneously, he experienced marked neurological deterioration characterized by a trend toward deep somnolence and hypoactivity, forcing the suspension of oral intake due to an imminent risk of aspiration and initiating total parenteral nutrition calculated at 2,200 kcal/24h.

Upon documentation of definitive high-flow failure and the progression of neurological decline toward a comatose state with a Glasgow Coma Scale score of 3/15 and the appearance of the Stenon-Louis ocular sign, an interdisciplinary medical consensus meeting was held with the immediate family nucleus. Taking into consideration the biological futility of invasive maneuvers, orotracheal intubation, cardiopulmonary resuscitation, and mechanical ventilation with high positive pressures were declined, proceeding to the legal execution of the advance directive document. The case was formally reviewed by the palliative medicine service, authorizing his transfer from intermediate care to the general hospitalization floor for the initiation of a programmed comfort palliative sedation protocol.

During his stay on the general medical ward, the patient remained in a state of deep stupor under controlled palliative sedation with a Glasgow Coma Scale score of 4/15, receiving continuous intravenous infusions of midazolam (15 mg/24h), morphine (8 mg/24h), and dexmedetomidine (400 µg), associated with a progressive weaning of oxygen flow directed toward comfort and a minimal hydric infusion of crystalloid solution at 10 mL/h to prevent fluid overload. Over the course of his evolution, the monitor demonstrated a total absence of blood pressure, and the loss of central pulses, inaudible heart sounds, and cessation of spontaneous respiratory movements were confirmed at the bedside. These findings were corroborated by a completely isoelectric electrocardiographic tracing on telemetry, and death was officially declared within the hospital facilities.

### III. METHODS

The methodological design employed consisted of an observational, descriptive, and retrospective analysis focused on the clinical, analytical, and ventilatory-palliative management of a geriatric patient with acute hypoxemic respiratory failure secondary to severe bacterial pneumonia. Clinical data collection was performed through an exhaustive review of the electronic health record, chronologically evaluating logs from the emergency department, the critical care unit, and the general hospitalization ward.

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For the monitoring of respiratory function and the prediction of success or failure of non-invasive ventilatory support, the ROX Index was implemented as a standard bedside predictive tool. This index was mathematically calculated using the formula that divides the peripheral oxygen saturation to fraction of inspired oxygen ratio ( $\text{SpO}_2/\text{FiO}_2$ ) by the patient's respiratory rate, recording its fluctuations against modifications in flow parameters.

Microbiological diagnosis and the exclusion of common viral or atypical bacterial pathogens were executed through a standardized nasopharyngeal swab obtained upon patient admission. The sample was processed using a molecular nucleic acid amplification test via multiplex polymerase chain reaction (PCR) (FilmArray), ensuring high-specificity screening within the first hours of care.

Systematic analytical monitoring included daily plasma quantification of acute phase reactants and organ function tests. C-reactive protein (CRP) levels were determined by immunoturbidimetry, procalcitonin via electrochemiluminescence, and cellular counts through automated flow cytometry, correlating their kinetics with the host's systemic clinical status.

Fluid management and metabolic support were structured according to body surface area and the severity of the infectious state. A methodological strategy of fluid restriction and pulmonary optimization was implemented through hourly input and output balances, complemented by the administration of loop diuretics and the initiation of Total Parenteral Nutrition (TPN) via a central venous line, strictly calculated to prevent tissue catabolism.

The process of limitation of life-sustaining treatment (LLST) was guided by the bioethical principles of beneficence and non-maleficence, coordinated by an interdisciplinary consensus medical committee. The clinical futility of implementing invasive high-positive pressure maneuvers or cardiopulmonary resuscitation was formally evaluated within the context of advanced senectud and diffuse alveolar damage, proceeding to the formal validation and legal signature of the regulatory advance directive document with the responsible family members.

Finally, the terminal palliative protocol for comfort and relief of physical suffering was scheduled through the use of continuous, titrated intravenous infusions of short-acting benzodiazepines and major opioids on the general medical floor. A controlled and progressive downward weaning of supplemental oxygen flows was executed, directed exclusively toward preventing extreme airway dryness and ensuring the control of refractory dyspnea during the dying phase.

## IV. RESULTS

A transient optimization of pulmonary function and gas exchange was observed during the first 24 hours of clinical monitoring under advanced oxygen therapy. Algorithmic

titration of high-flow nasal cannula parameters to a flow rate of 50 L/min with a fraction of inspired oxygen at 33% achieved an elevation of peripheral oxygen saturation to 94%, reaching a calculated ROX Index of 13.5, which confirmed adequate carbon dioxide clearance and initial alveolar recruitment without requiring vasopressor support.

Respiratory molecular screening via the FilmArray panel yielded negative results for all 20 pathogens on the platform, ruling out co-infections by influenza, respiratory syncytial virus, or atypical bacteria. Furthermore, bilateral blood cultures drawn upon admission reported no microbiological growth at 48 hours of incubation, isolating the infectious focus strictly to the bilateral pulmonary parenchyma and confirming an inflammatory response of localized pulmonary origin without concomitant bacteremia.

Serial hematological analyses demonstrated a reactive leukocytosis with steadily increasing neutrophilia, progressing from an initial count of  $10.52 \times 10^3/\mu\text{L}$  to a maximum peak of  $11.79 \times 10^3/\mu\text{L}$  on the third day of critical care stay, with neutrophils at  $9.79 \times 10^3/\mu\text{L}$ . Acute phase reactants showed a moderate downward trend on the fourth day of hospitalization, recording a C-reactive protein of 6.29 mg/dL and a procalcitonin of 0.17 ng/mL; however, persistent refractory moderate thrombocytopenia was noted, with platelet counts fluctuating between  $69 \times 10^3/\mu\text{L}$  and  $89 \times 10^3/\mu\text{L}$  throughout the entire evolution.

Clinical ventilatory monitoring confirmed the definitive failure of non-invasive support on the third day after admission, manifesting a severe increase in work of breathing, shallow polypnea, and erratic tachypnea with rates of 16 to 30 rpm. Despite elevating high-flow nasal cannula parameters to their maximum capacity with a flow rate of 60 L/min and a fraction of inspired oxygen at 60%, the patient maintained a borderline oxygen saturation of 91% with extreme difficulty, formally consolidating clinical and imaging criteria for severe acute respiratory distress syndrome with absolute refractory hypoxemia on the following day.

Neurological function experienced a progressive and irreversible collapse secondary to diffuse tissue hypoxemia and the severity of the evolving pulmonary septic shock. The patient transitioned from a state of somnolence and hypoactivity to a deep coma on the fourth day of critical care stay, registering a score of 3/15 on the Glasgow Coma Scale. This deterioration clinically coincided with the appearance of advanced signs of irreversible cephalic hypoperfusion and cellular dehydration, highlighting the objective presence of the Stenon-Louis ocular sign.

The protocolized transition to the general medical ward under the framework of limitation of life-sustaining treatment permitted the successful withdrawal of redundant invasive support measures. The remaining total parenteral nutrition was definitively discontinued, and intravenous fluid inputs were reduced to a minimum maintenance infusion of

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crystalloid solution at 10 mL/h. This strict fluid and electrolyte adjustment effectively prevented the development of additive volume overload, systemic congestion, and the onset of terminal respiratory secretions (death rattles) audible at a distance.

The results of continuous intravenous palliative sedation demonstrated optimal symptom control during the dying phase and final outcome. Combined infusions of midazolam at a rate of 15 mg every 24 hours and morphine at a rate of 8 mg every 24 hours, alongside the adjuvant use of dexmedetomidine (400 µg), completely abolished macroscopic signs of dyspnea and psychomotor agitation, maintaining the patient in a state of comfortable stupor until clinical cardiorespiratory arrest and a completely isoelectric electrocardiographic tracing on telemetry were documented, with death officially declared within the hospital facilities.

### V. DISCUSSION

The clinical management of severe community-acquired pneumonia complicated by acute respiratory distress syndrome (ARDS) in the extreme geriatric population imposes massive biological, ethical, and healthcare challenges for contemporary critical care medicine. Immunological senescence, or immunosenescence, irreversibly alters intracellular signaling pathways, antigen presentation, and local neutrophilic chemotaxis, attenuating the baseline bactericidal capacity of alveolar macrophages, which explains the masked and atypical clinical presentations in this age group.<sup>1</sup>

In the present case, chronic exposure for over three decades to biomass smoke from wood combustion acted as a determining factor for structural lung vulnerability. Continuous inhalation of suspended particulate matter generates a low-grade inflammatory state and diffuse interstitial fibrosis that destroys normal ciliary architecture and perpetuates mucociliary clearance dysfunction, facilitating the rapid progression of an acute infectious insult into a bilateral consolidation syndrome and refractory respiratory failure.<sup>2</sup>

From a differential diagnostic perspective, it is crucial to distinguish the clinical manifestations intrinsic to CAP from the onset of ARDS. While CAP is initially characterized by symptoms of localized infectious and parenchymal involvement—such as fever, productive cough, pleuritic chest pain, and a localized consolidation syndrome with inspiratory crackles—progression toward ARDS manifests as acutely onset and rapidly progressive dyspnea, extreme tachypnea, frank use of accessory musculature, and severe hypoxemia refractory to oxygen therapy, accompanied by diffuse bilateral alveolar infiltrates that extend beyond the initial lobar involvement.<sup>3</sup>

The rapid onset and progression toward severe ARDS exemplifies the destructive cascade triggered by pulmonary-source sepsis. Diffuse alveolar damage critically

compromises gas exchange due to the filling of air spaces with a proteinaceous inflammatory exudate, generating massive right-to-left intrapulmonary shunts and explaining the refractory hypoxemia observed despite the optimization of advanced oxygen therapy devices.<sup>4</sup>

This fulminant evolution necessitates a strict evaluation of CAP severity criteria at the patient's bedside, validating a poor short-term prognosis and the relevance of bioethical decision-making. To objectify the severity of the clinical picture upon admission, justify the initial need for critical monitoring, and substantiate the futility of subsequent invasive measures, the CURB-65, SMART-COP, and ATS/IDSA severity criteria scales were utilized complementarily (see Tables 1, 2, and 3).<sup>5</sup>

Clinically, the patient's course unequivocally met validated international minor and major severity criteria (such as ATS/IDSA and CURB-65/PSI scales). Key minor criteria included frank tachypnea (respiratory rate  $\geq 30$  rpm), relative hypothermia, confusion/somnolence secondary to cerebral hypoperfusion, laboratory-documented moderate thrombocytopenia, and bilateral multilobar radiological involvement. Furthermore, absolute respiratory failure leading to the need for advanced ventilatory support represented the fulfillment of a major criterion, confirming an unfavorable short-term prognosis and the appropriateness of bioethical decision-making.<sup>5</sup>

A critical factor that worsened the patient's clinical course was iatrogenic immunosuppression secondary to prior administration of systemic corticosteroids that were abruptly discontinued. Prolonged corticosteroid therapy alters the transcription of essential cytokines, hindering the eradication of the initial pathogen and elevating the risk of superinfection by multidrug-resistant or opportunistic organisms such as *Pneumocystis jirovecii*, which methodologically justified the initiation of empirical broad-spectrum therapies combined with targeted agents.<sup>6</sup>

The introduction of post-pandemic clinical practice guidelines highlights continuous and non-invasive oxygenation monitoring via the ROX Index as a fundamental pillar at the bedside of patients receiving support with high-flow nasal cannulas. Close monitoring of the relationship between oxygen saturation and inspired fraction relative to respiratory rate allows for the early discrimination of imminent non-invasive therapy failure, avoiding decision delays and mitigating patient-self-induced lung injury due to excessive work of breathing.<sup>7</sup>

The definitive refractory failure of supplemental oxygen therapy raises a profound ethical dilemma regarding the use of invasive high-positive pressure ventilatory interventions in the ninth decade of life. The biotrauma and biomechanical complications inherent to orotracheal intubation in frail elderly patients with established severe ARDS lack real clinical benefit, configuring a clear scenario of therapeutic futility and necessitating the reorientation of medical

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objectives under the bioethical concepts of beneficence and non-maleficence.<sup>7</sup>

In this context of extreme gravity and respiratory biological futility, the legal framework conferred by an advance directive document permits the execution of limitation of life-sustaining treatment to safeguard patient dignity. The consensual withdrawal of futile mechanical or artificial metabolic supports, such as total parenteral nutrition, constitutes a medical act of high scientific and human competence, redirecting clinical practice exclusively toward intensive palliative comfort care at the end of life.<sup>6,7</sup>

The implementation of an optimal palliative regimen requires meticulous and assertive pharmacological titration to neutralize the distress derived from acute respiratory failure. Combined infusions of short-acting benzodiazepines and potent opioids such as morphine act synergistically within the central nervous system, modulating the perception of refractory dyspnea and mitigating passive pain without inducing an artificial prolongation of the dying process.<sup>7</sup>

Additionally, the methodological control of fluid balances during the terminal comfort support phase is a critical intervention to prevent distressing symptoms. Drastic restriction of maintenance solutions to minimal rates prevents the elevation of capillary hydrostatic pressure in a previously injured lung parenchyma, categorically hindering fluid accumulation and the consequent appearance of terminal respiratory secretions (death rattles) that are highly distressing to the family nucleus.<sup>7</sup>

Finally, new global consensus guidelines on post-pandemic respiratory distress emphasize the need to expand traditional definitions to incorporate global clinical and socioeconomic heterogeneity. By validating non-invasive diagnostic tools such as lung ultrasound and peripheral saturation indices, critical care medicine transitions toward a more equitable and dynamic construct that optimizes daily prognostic judgment by precisely delimiting futile interventions from truly proportional therapeutics.<sup>7</sup>

Table 1. CURB-65 Severity Score and Risk Stratification

| Criterion | Clinical / Laboratory Feature | Cut-off Value                  | Patient Finding & Score                      |
|-----------|-------------------------------|--------------------------------|----------------------------------------------|
| C         | Confusion                     | New-onset acute disorientation | Present (Somnolence/Stupor) → 1 pt           |
| U         | Urea                          | >7 mmol/L (>19 mg/dL)          | Pending correlation → 0 pt                   |
| R         | Respiratory Rate              | ≥30 breaths/min                | Present (32 breaths/min at admission) → 1 pt |

|       |                          |                                         |                                                                       |
|-------|--------------------------|-----------------------------------------|-----------------------------------------------------------------------|
| B     | Blood Pressure           | Systolic <90 mmHg or Diastolic ≤60 mmHg | Absent (130/86 mmHg) → 0 pt                                           |
| 65    | 65 years of age or older | ≥65 years                               | Present (92 years old) → 1 pt                                         |
| Total | Cumulative Score         | ≥3 points = High Risk                   | 3 points (High risk, indicated for critical monitoring/ICU admission) |

Table 2. ATS/IDSA Severity Criteria for Severe Community-Acquired Pneumonia (CAP)

| Category                                | Validated Guidelines Criteria                                                                                                                      | Patient-Specific Clinical Status                                                                                                                                               | Evaluation                                                                                       |
|-----------------------------------------|----------------------------------------------------------------------------------------------------------------------------------------------------|--------------------------------------------------------------------------------------------------------------------------------------------------------------------------------|--------------------------------------------------------------------------------------------------|
| Major Criteria (Presence of at least 1) | <ul style="list-style-type: none"><li>Invasive mechanical ventilation requirement</li><li>Septic shock necessitating vasopressor support</li></ul> | <ul style="list-style-type: none"><li>Failed non-invasive high-flow support; neurological collapse.</li><li>Refractory hypoxemia with absolute tissue hypoperfusion.</li></ul> | Met (Invasive maneuvers declined due to advanced clinical futility and signed Advance Directive) |

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|                                    |                                                                                                                                                                                                                                                                                                                                                                                                                                                              |  |  |
|------------------------------------|--------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------|--|--|
| Minor Criteria<br>(Presence of ≥3) | 1. Respiratory Rate: $\geq 30$ breaths/min<br>2. PaO <sub>2</sub> /FiO <sub>2</sub> ratio: $\leq 250$<br>3. Multilobar infiltrates: Bilateral involvement<br>4. Confusion/Di orientation: Acute onset<br>5. Uremia: BUN $\geq 20$ mg/dL<br>6. Leukopenia: WBC $< 4,000$ cells/ $\mu$ L<br>7. Thrombocytopenia: Platelets $< 100,000$ / $\mu$ L<br>8. Hypothermia: Core temp $< 36^\circ\text{C}$<br>9. Hypotension: Requiring aggressive fluid resuscitation |  |  |
|                                    |                                                                                                                                                                                                                                                                                                                                                                                                                                                              |  |  |

|       |                  |                                                              |                                                                                            |
|-------|------------------|--------------------------------------------------------------|--------------------------------------------------------------------------------------------|
| T     | Tachycardia      | $\geq 125$ beats/min                                         | Absent (85 bpm at admission) → 0 pt                                                        |
| C     | Confusion        | New-onset acute confusion                                    | Absent at initial triage (GCS 15/15) → 0 pt                                                |
| O     | Oxygenation      | SpO <sub>2</sub> $\leq 93\%$ or PaO <sub>2</sub> $< 70$ mmHg | Present (SpO <sub>2</sub> 78% on room air) → 2 pt                                          |
| P     | Ph               | Arterial pH $< 7.35$                                         | Pending correlation → 0 pt                                                                 |
| Total | Cumulative Score | $\geq 3$ points = High Risk                                  | 4 points (High risk for requiring advanced ventilatory support or critical care admission) |

## CONCLUSION

The present clinical case highlights the significance of merging critical care criteria with a bioethically solid geriatric and palliative approach when managing patients in their ninth decade of life. In this scenario, successful clinical judgment lies in discerning the patient's semiological transition; identifying that the persistence of infectious symptomatology in CAP and the fulfillment of its multifactorial severity criteria—such as tachypnea, acute confusion, and thrombocytopenia—are not isolated events, but rather the prelude to severe ARDS characterized by diffuse alveolar collapse and refractory hypoxemia.

Severe community-acquired pneumonia complicated by septic shock and severe acute respiratory distress syndrome carries an inherently ominous prognosis in frail elderly individuals, where the optimization of non-invasive supports offers a clinically valuable window for close monitoring and multidisciplinary deliberation, but under no circumstances should it delay definitive decisions regarding therapeutic limits in the face of frank biological futility.

The limitation of life-sustaining treatment, formally backed by legal instruments such as advance directive documents, constitutes a medical act of high scientific, ethical, and human competence. By withdrawing futile artificial metabolic supports such as total parenteral nutrition and guiding the dying phase through continuous and titrated infusions of benzodiazepines and major opioids associated with strict maintenance fluid restriction, contemporary intensive

**Table 3. SMART-COP Scoring System for Predicting Intensive Respiratory or Vasopressor Support.**

| Acronym | Clinical / Laboratory Criterion | Cut-off Value             | Patient Finding & Score                      |
|---------|---------------------------------|---------------------------|----------------------------------------------|
| S       | Systolic Blood Pressure         | $< 90$ mmHg               | Absent (130 mmHg) → 0 pt                     |
| M       | Multilobar infiltrates          | Present on Chest X-ray/CT | Present (Bilateral consolidation) → 1 pt     |
| A       | Albumin                         | $< 3.5$ g/dL              | Pending correlation → 0 pt                   |
| R       | Respiratory Rate                | $\geq 30$ breaths/min     | Present (32 breaths/min at admission) → 1 pt |

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medicine successfully transitions from advanced organ support to the absolute relief of respiratory suffering, guaranteeing a peaceful and humanized environment.

From a methodological and healthcare safety perspective, the implementation of the ROX Index at the bedside of the frail geriatric patient establishes itself as the non-invasive gold standard to guide therapeutic proportionality in real time. By dynamically correlating ventilatory dynamics with peripheral oxygenation goals, this tool objectively identifies the inflection point where high-flow support ceases to be beneficial and becomes a promoter of self-induced lung injury, marking the exact moment to suspend futile measures and transition to exclusive comfort protocols.

Likewise, the success of the terminal palliative approach in a previously injured lung parenchyma depends critically on a strict restrictive control of intravenous fluid infusion. Scheduling crystalloid solutions reduced to the minimum basal biological maintenance prevents an increase in pulmonary capillary hydrostatic pressure across an alveolar-capillary barrier with previously altered permeability, effectively intervening to block fluid congestion and categorically abolish the accumulation of secretions and the consequent onset of terminal respiratory secretions (death rattles) that cause distress to the family nucleus.

Finally, the adoption of new guidelines and global consensus statements on post-pandemic respiratory distress imposes the need to re-evaluate daily intensive medical practice in favor of more equitable and humane pathways. The recognition that acute diffuse respiratory syndrome can be validly diagnosed and staged using non-invasive clinical and imaging markers opens a new era toward precision and personalized medicine, where preserving the intrinsic dignity of the terminally ill, critical geriatric patient during the dying phase is as high a priority as pursuing survival in patients with a recoverable prognosis.

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