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Nutritional Challenges in ARDS Secondary to Pneumonoultramicroscopicsilicovolcanoconiosis: A Case Report

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Abstract

Acute Respiratory Distress Syndrome (ARDS) is a critical condition associated with high morbidity and mortality, particularly when accompanied by chronic lung disease and multiple organ dysfunction. Nutritional management in patients with ARDS remains challenging due to increased metabolic demands, reduced oral intake capacity, and complications such as acute kidney injury (AKI). This challenge becomes more complex in patients with *pneumonoultramicroscopicsilicovolcanoconiosis*, a rare occupational lung disease caused by prolonged exposure to fine silica particles. This study aimed to describe the challenges of nutritional management in a patient with ARDS secondary to *pneumonoultramicroscopicsilicovolcanoconiosis* and to highlight the importance of individualized nutritional support in critically ill patients. This study employed a descriptive case report approach. Data were obtained from clinical observations, medical records, nutritional assessments, laboratory examinations, and treatment progress of a 38-year-old male mine worker with severe ARDS, suspected pneumoconiosis, and AKI who was treated in the intensive care unit. The patient experienced difficulties in meeting nutritional requirements due to persistent dyspnea, nausea, decreased appetite, increased oxygen requirements, and renal dysfunction. Nutritional therapy was adjusted according to clinical changes, beginning with an oral soft diet and subsequently modified to a non-dialysis kidney diet with protein restriction. Despite multidisciplinary management involving respiratory support, nutritional intervention, and renal monitoring, the patient's condition deteriorated and required invasive mechanical ventilation. Nutritional management in patients with ARDS complicated by occupational lung disease and organ dysfunction requires continuous evaluation and individualized strategies. Further studies are needed to establish evidence-based nutritional approaches for this complex patient population.

INTRODUCTION

Acute Respiratory Distress Syndrome (ARDS) is a form of acute respiratory failure characterized by with hypoxemia heavy, infiltrate bilateral on lungs, And edema lungs noncardiogenic due to the increase alveolo-capillary membrane permeability. ARDS Still become Wrong One reason major morbidity and mortality on patient critical, with numbers

mortality range 30–45%, depends to the degree severity and disease Which underlying cause. Current ARDS management focuses on supportive therapy, including mechanical ventilation protective, prone position, and optimization of therapy for the underlying cause (Qadir et al., 2024; Grasselli et al., 2023).

Besides therapy respiration, therapy nutrition is an integral part of the management of ARDS patients. Condition hypermetabolic and hypercatabolic which occurs as a result of a systemic inflammatory response causing improvement need energy and protein, so that patient risky tall experience malnutrition, loss of muscle mass, and worse clinical outcomes. Current guidelines recommend giving nutrition enteral early if there are no contraindications, with the aim of maintaining the integrity of the intestinal mucosa, reducing infectious complications, and supporting the healing process (Qadir et al., 2024; Grasselli et al., 2023).

Recent studies have strengthened the evidence regarding the relationship between silica dust exposure and impaired respiratory function, which may increase susceptibility to severe respiratory failure. Batool et al. (2025), through a study involving mine workers, demonstrated an association between silica dust exposure and pulmonary dysfunction, emphasizing the importance of occupational exposure history in assessing respiratory diseases among mining populations. Mundt et al. (2025), through a systematic review, further evaluated the dose–response relationship between respirable crystalline silica exposure and silicosis as well as lung cancer, reinforcing evidence that exposure intensity and duration are important determinants in the development of silica-related pulmonary diseases. Meanwhile, specific studies on pneumonoultramicroscopicsilicovolcanoconiosis remain extremely limited. An earlier report by Trisnawati et al. described this condition as a fibrotic parenchymal lung disease associated with chronic inhalation of inorganic dust containing crystalline silicon dioxide. These findings indicate that pulmonary disorders caused by dust exposure represent chronic pathological processes that may increase vulnerability when patients experience pneumonia, systemic inflammation, and ARDS (Chavda et al., 2025; Comunian et al., 2020; Huang et al., 2025).

On the other hand, advances in nutritional research among critically ill patients indicate that nutritional therapy can no longer be considered merely supportive treatment but must be adjusted according to disease phase, catabolic level, organ function, and metabolic tolerance. Heyland et al. (2023), through the EFFORT Protein Trial involving 1,301 patients from 85 ICUs across 16 countries, found that high-dose protein administration did not accelerate hospital discharge alive compared with standard protein administration. Furthermore, high protein intake showed potential unfavorable effects among patients with acute kidney injury (AKI) and more severe organ failure. This finding is highly relevant to the present study because the patient experienced not only severe ARDS but also AKI, requiring nutritional adjustment into a non-dialysis renal diet with protein intake of 0.8 g/kg body weight/day and energy requirements of approximately 30 kcal/kg body weight/day. Di Mario et al. (2025) also emphasized that AKI in critically ill patients may be accompanied by protein-energy wasting; therefore, energy requirements, protein targets, timing of administration, and nutritional routes should be determined individually.

The complexity of nutritional therapy becomes increasingly significant when ARDS is accompanied by mechanical ventilation, sepsis, renal dysfunction, decreased consciousness, and feeding intolerance. Contemporary evidence suggests that aggressive nutritional approaches during the early phase of critical illness do not necessarily produce better clinical

outcomes. Studies regarding enteral nutrition in critically ill patients indicate that a more restrictive nutritional approach during the acute phase may be more appropriate than immediate full-target feeding. Similarly, high-dose protein administration has not demonstrated clear superiority and may potentially be harmful in certain populations, particularly patients with AKI. Furthermore, Wu and Chasteen (2024) reported that ventilator-induced diaphragmatic dysfunction may develop within hours to several days after mechanical ventilation initiation, contributing to respiratory muscle weakness and complicating ventilator weaning processes. The relationship between nutritional status and ARDS prognosis has also received increasing attention. Zhao et al. (2026), in a cohort study involving 1,022 ICU patients with sepsis-associated ARDS, found that the Prognostic Nutritional Index was independently associated with 28-day mortality and improved prognostic stratification when combined with SOFA and APACHE II scores. These findings demonstrate that nutrition, organ dysfunction, and respiratory support are interconnected components influencing the clinical trajectory of ARDS patients (Tang et al., 2026; Zhou et al., 2025).

Although research on pneumoconiosis, ARDS, mechanical ventilation, AKI, and nutritional therapy in critically ill patients has continued to develop, most previous studies have examined these conditions separately. The literature on silicosis has primarily focused on occupational exposure, fibrosis pathogenesis, pulmonary function decline, and chronic respiratory outcomes, whereas nutritional studies in critically ill patients generally involve heterogeneous ICU populations without specific emphasis on ARDS occurring in patients with occupational dust-related lung disease. Research on protein dosing in critically ill patients has also demonstrated important interactions with AKI; however, limited evidence explains how nutritional decisions should be adapted when AKI occurs simultaneously with severe respiratory failure in patients with fibrotic lung disease caused by chronic dust exposure. More specifically, clinical reports regarding nutritional challenges in ARDS associated with pneumonoultramicroscopicsilicovolcanoconiosis remain scarce. This research gap highlights the need for case reports integrating respiratory disease progression, renal dysfunction, changes in nutritional routes and targets, ventilation requirements, and clinical challenges encountered during intensive care management.

The urgency of this study lies in the need to describe real-world nutritional decision-making in patients with complex critical illness that cannot be fully explained through nutritional recommendations for individual diseases alone. In the reported case, nutritional fulfillment was challenged by persistent dyspnea, nausea, reduced appetite, increased oxygen requirements, and the development of AKI, which limited the patient's ability to meet nutritional needs through oral intake. The respiratory condition subsequently fluctuated and deteriorated, with persistent severe hypoxemia despite maximal oxygen supplementation, followed by the requirement for non-invasive ventilation and eventually invasive mechanical ventilation. The novelty of this case report lies in its integrated description of nutritional therapy challenges throughout the course of severe ARDS in a patient with suspected pneumoconiosis due to occupational dust exposure, complicated by AKI, severe infection, progressive respiratory support requirements, and limitations in intervention due to patient and family decisions. This approach provides a different clinical perspective from previous studies that generally evaluate silica-related lung disease, ARDS, AKI, or nutritional interventions separately.

Based on this research gap, this study aimed to describe the challenges of nutritional management in patients with ARDS associated with pneumonoultramicroscopicsilicovolcanoconiosis, particularly in relation to changes in respiratory status, the development of AKI, limitations in oral intake, adjustment of energy and protein requirements, and disease progression during intensive care treatment. Academically, this case report is expected to contribute to the literature regarding the relationship between occupational lung disease, ARDS, multiorgan dysfunction, and nutritional therapy in critically ill patients. Clinically, the findings may increase healthcare professionals' awareness regarding the importance of dynamic and individualized nutritional evaluation in ARDS patients, especially those with renal impairment and progressive ventilatory support requirements. Furthermore, this study may provide consideration for intensivists, pulmonologists, nephrologists, and clinical nutritionists in developing multidisciplinary approaches to balance energy and protein requirements, nutritional tolerance, renal function, and respiratory demands, allowing therapeutic decisions to be more appropriately adapted to changes in patients' clinical conditions.

METHOD

A 38-year-old man, a mine worker with five years of occupational experience and a history of exposure to smoke and dust in the workplace environment, was admitted to the intensive care unit on April 5, 2026. The patient presented with a chief complaint of progressive shortness of breath that had been experienced for four days prior to hospital admission and had worsened on the day of admission. The complaint was accompanied by a cough that had started one day before hospitalization without fever. The patient also reported epigastric pain accompanied by one episode of vomiting and nausea, which had resolved at the time of initial assessment, without associated chest pain. The patient denied any previous medical history and reported undergoing regular periodic health examinations through his workplace without significant abnormalities. A history of active smoking for 20 years was also noted as an additional occupational and lifestyle-related risk factor.

On initial examination after receiving oxygen supplementation via a non-rebreather mask (NRM) at 15 liters/minute, the patient appeared moderately ill but remained conscious and alert (*compos mentis*) (GCS E4M6V5). Vital signs showed a blood pressure of 134/78 mmHg, pulse rate of 75 beats/minute, respiratory rate of 30 breaths/minute, temperature of 36.6°C, and peripheral oxygen saturation of 88–91% despite maximum oxygen supplementation. Thoracic examination revealed decreased vesicular breath sounds in the right hemithorax, bilateral coarse rhonchi, and prolonged expiration without wheezing. Abdominal examination was within normal limits, while peripheral edema was observed in the extremities with adequate peripheral perfusion.

Initial arterial blood gas analysis showed pH 7.35, pCO_2 32 mmHg, pO_2 66 mmHg, HCO_3^- 19 mmol/L, and SaO_2 92%, consistent with hypoxemia accompanied by partially compensated respiratory alkalosis; however, repeat examination on the same day showed deterioration to pH 7.31 with HCO_3^- 17 mmol/L, indicating the development of a metabolic acidosis component. Complete blood count demonstrated leukocytosis (20,960/µL), with hemoglobin, hematocrit, and platelet levels within normal limits. Renal function tests showed elevated urea (215 mg/dL) and creatinine (10.8 mg/dL), accompanied by mild hyponatremia

and hypokalemia (sodium 132 mmol/L, potassium 3.4 mmol/L, chloride 94 mmol/L). Chest radiography revealed an elevated right hemidiaphragm with suspicion of pleural effusion versus passive atelectasis, along with increased bilateral bronchovascular markings. However, bedside thoracic ultrasonography revealed no pleural effusion, and inferior vena cava assessment indicated adequate intravascular volume status.

Based on these findings, the patient was diagnosed with severe acute respiratory distress syndrome (ARDS), interstitial pneumonia suspected to be caused by bacterial infection with differential diagnoses of pneumoconiosis and viral infection, severe community-acquired pneumonia (CURB-65 score: 2), acute exacerbation of chronic obstructive pulmonary disease (COPD), acute kidney injury (AKI) stage 3, as well as hypokalemia and hyponatremia.

Initial management included non-invasive ventilation with a target oxygen saturation of 88–92%, crystalloid fluid resuscitation, and empirical antibiotic therapy consisting of ceftriaxone 2 g/24 hours and levofloxacin 500 mg/24 hours intravenously. The patient also received dexamethasone 5 mg/12 hours with a tapering plan, inhaled ipratropium-salbutamol bronchodilator therapy, N-acetylcysteine, and ranitidine. During the first four days of treatment, partial clinical improvement was observed, marked by a reduction in oxygen requirements from NRM 15 liters/minute to 10 liters/minute, with oxygen saturation improving to 93–95%. However, renal function did not demonstrate similar improvement. The recommendation for hemodialysis from the nephrology consultant was refused by the patient and family; therefore, conservative management consisting of folic acid supplementation, vitamin B12 administration, and avoidance of nephrotoxic agents was continued. HIV testing during this period showed non-reactive results.

Nutritional therapy was provided as part of the management strategy while the patient was being treated in the intensive care unit. The patient had a body weight of 65 kg and a height of 167 cm (BMI 23.3 kg/m²). Based on nutritional requirement calculations, the estimated energy requirement was 1,539.3 kcal/day.

During the initial treatment phase, while the patient was still able to tolerate oral intake, a soft diet providing 2,100 kcal/day was administered, along with a target oral fluid intake of 1,500 mL/day. During hospitalization, meeting nutritional requirements became increasingly challenging because the patient experienced persistent dyspnea, nausea, decreased appetite, and increased oxygen requirements, which limited the ability to maintain adequate oral intake.

As the disease progressed, the patient developed AKI; therefore, nutritional management was adjusted according to recommendations from the Nephrology Division in the form of a non-dialysis renal diet with a protein intake of 0.8 g/kg body weight/day and energy requirements of 30 kcal/kg body weight/day. This intervention was accompanied by strict monitoring of urine output, fluid balance, and avoidance of nephrotoxic medications.

Nutritional monitoring was performed periodically, including evaluation of energy requirements, maintenance of the head-of-bed position at 30°, repositioning every two hours, blood glucose monitoring every 12 hours, and supportive gastrointestinal therapy consisting of sucralfate and lactulose.

From the tenth to the fifteenth day of hospitalization, respiratory status fluctuated, with oxygen saturation ranging from 78–92% despite maximum oxygen supplementation, accompanied by orthopnea. A non-contrast chest CT scan performed on April 12, 2026, demonstrated severe bilateral pneumonia suggestive of bacterial etiology. Persistent

leukocytosis (25,000–26,000/μL) raised suspicion of sepsis with possible cytokine storm; therefore, antibiotic therapy was escalated to meropenem (dose adjusted according to renal function) combined with fluconazole. Renal function was assessed as AKI stage 3 with a differential diagnosis of acute-on-chronic kidney disease. The family again refused the recommendation for hemodialysis. The differential diagnosis was expanded to include *Pneumocystis* pneumonia and viral infections. SARS-CoV-2 PCR testing was planned but could not be performed at the local facility, and referral to a more comprehensive healthcare facility was recommended but declined by the family.

From the sixteenth to the nineteenth day of treatment, renal function showed partial improvement, with creatinine levels decreasing to 1.9–2.9 mg/dL. However, this improvement was accompanied by worsening respiratory status. Oxygenation support was escalated to non-invasive ventilation with FiO_2 100% and PEEP 8 cmH_2O, without significant improvement in oxygen saturation (80–82%), accompanied by decreased consciousness to an apathetic state. Clinical suspicion during this phase included refractory ARDS, sepsis due to bacterial or fungal pneumonia with possible cytokine storm, COPD exacerbation, and pulmonary embolism or microemboli in the context of sepsis. The family was informed regarding the possible requirement for invasive mechanical ventilation and agreed to intubation, central venous catheter placement, and cardiopulmonary resuscitation if clinically indicated.

On April 20, 2026, the patient underwent endotracheal intubation and invasive mechanical ventilation initiation with an initial V-SIMV mode and FiO_2 of 100%, which was subsequently switched to P-SIMV mode. Continuous propofol and fentanyl sedation-analgesia were administered, along with low-dose vasoactive support. Antibiotic therapy was further escalated to meropenem 1 g/8 hours, with additional methylprednisolone 62.5 mg/8 hours and supportive therapies including N-acetylcysteine, sucralfate, lactulose, vitamin D, and sodium bicarbonate. Although the patient initially remained hemodynamically stable without vasopressor support, the pulse rate progressively increased to 132–152 beats/minute, with persistently low oxygen saturation (73–75%) despite maintaining FiO_2 at 100%. Further discussions with the family regarding the worsening clinical condition resulted in an agreement to decline repeated cardiopulmonary resuscitation while continuing maximal medical therapy.

At 22:39 Western Indonesian Time (WIT), the patient experienced sudden deterioration characterized by loss of response, GCS E1M1Vt, and severe bradycardia (pulse rate 32 beats/minute) accompanied by unmeasurable hemodynamic parameters. At 22:45 WIT, clinical signs of cardiac arrest were identified, including bilateral fixed mydriasis (7 mm/7 mm) without pupillary light reflex and corneal reflex, absence of heart sounds and breath sounds, cold extremities, capillary refill time exceeding three seconds, and an electrocardiographic finding of asystole. In accordance with the previous agreement, aggressive resuscitation was not performed; however, atropine sulfate 1 mg was administered as part of maximal medical management. The patient was declared deceased at 22:46 WIT in the presence of his wife, and the endotracheal tube was removed following confirmation of death.

The final diagnosis was respiratory failure requiring mechanical ventilation due to severe ARDS without improvement, sepsis secondary to pneumonia with suspected cytokine storm, interstitial pneumonia suspected to be caused by bacterial infection with differential diagnoses of pneumoconiosis, viral infection, and *Pneumocystis* pneumonia, severe community-acquired pneumonia (CURB-65 score: 2), acute exacerbation of COPD secondary

to pneumonia with chronic hypoxemia, AKI stage 2 with differential diagnosis of acute-on-chronic kidney disease, and electrolyte disturbances including hypokalemia.

RESULTS AND DISCUSSION

Process Pneumoconiosis is disease group lungs interstitial arise consequence chronic inhalation particle dust inorganic in environment work, which on worker mine generally in the form of silica dust crystalline, rock coal, or mixture mineral other Which trapped in the parenchyma lungs and triggers a chronic inflammatory response and progressive fibrosis that is essentially irreversible even after exposure has been discontinued (Hua et al., 2023; Hou et al., 2025). Chronic exposure to submicron-sized particles allows deposition until to unit terminal respiratory, in where process phagocytosis by Alveolar macrophages trigger an inflammatory pathway involving autophagy, apoptosis, and proptosis cell epithelium and macrophages, Which on Finally move continuous inflammatory-fibrosis cycle in the lung parenchyma (Vanka et al., 2022). On in this case, work history as a miner during five year accompanied by exposure smoke and dust, plus history smoke active over twenty years, forming a synergistic combination of occupational and lifestyle risk factors speed up damage architecture lungs and reduce the patient's functional respiratory reserve, so that when an acute insult occurs in the form of an infection pneumonia, compensation capacity lungs which is already burdened by chronic fibrotic changes becomes very limited.

Acute Respiratory Distress Syndrome Which Originally established based on the 2012 Berlin definition, it has now been updated through the 2023–2024 global definition consensus which expands the diagnostic criteria by participate accommodate patient Which get therapy oxygen flow tall and non-invasive ventilation, the use of the peripheral saturation ratio to FiO2 (SpO2/FiO2) as alternative PaO2/FiO2 ratio, as well as utilization ultrasound thorax as an additional imaging modality, particularly for application at facilities with limited resources (Matthay et al., 2024; Grasselli et al., 2023). Patho physiologically, ARDS progresses through an exudative phase with breakdown of the alveolar-capillary barrier and leakage of protein-rich fluid into the space. alveolar, followed by the phase proliferative and on case prolonged fibrotic phase, a sequence that forms the basis of protective ventilation strategies and internal organ support order like severe ARDS (Grasselli et al., 2023). In this case, the picture hypoxemia heavy Which refractory to supplementation oxygen maximum, infiltrate bilateral on thoracic imaging, as well as journey clinical progressive worsening although has get non-invasive ventilation management eventually requiring invasive mechanical ventilation, consistent with severe ARDS likely due to a chronic fibrotic process due to underlying pneumoconiosis, with pneumonia bacteria as a factor acute trigger.

Condition critical like ARDS trigger hypermetabolic state and hypercatabolic Which marked by the imbalance between synthesis and muscle protein breakdown, at the rate of catabolism Which far beyond anabolism rate, so that patient critical can lost more than five twelve percent muscle mass only in One Sunday First intensive care, especially on patient with multiorgan failure (Fazzini et al., 2023). This condition is exacerbated by the prolonged use of invasive mechanical ventilation, which causes ventilator-induced diaphragmatic dysfunction (VIDD) through mitochondrial oxidative stress mechanisms and activation of the proteolysis pathway on fibre muscle diaphragm Which can start to occur in twelve to two twenty four hours First usage ventilator, thus prolonging dependence on ventilators and make things

difficult process weaning (Fu et al., 2025; Grasselli et al., 2023; Matthay et al., 2024). On case this, a long course of the disease for more than of twenty days of intensive care with dependency mechanical ventilation invasive since the second day tens, accompanied by leucocytosis persistent which indicates a sustained systemic inflammatory response until suspected existence storm cytokines, describing burden catabolic cumulative so that adequate nutritional support is an essential component in efforts to maintain muscle mass, immune function, and potential. recovery patient.

Determination need energy on patient ideally critical use calorimetry indirect as standard gold Because capable measure actual resting energy expenditure based on exchange gas, however limitations availability tool the in Lots facility health, included in order like case This, make predictive equation based heavy body as alternative commonly used although potential create bias estimate. Guide practical European Society for Clinical Nutrition and Metabolism (ESPEN) 2023 and American Society for Parenteral and Enteral Nutrition (ASPEN)/ Society of Critical Care Medicine (SCCM) 2022 recommend giving energy gradually in phase I beginning disease critical, with a less aggressive initial target to avoid overfeeding Which can increase carbon dioxide production and respiratory workload, then increase towards the requirement full along stabilization phase I (Singer et al., 2023; Compher et al., 2022). On case This, need energy counted as much as 1.539.3 kcal/day Which equivalent with around 23.7 kcal/kgBW/day based on a body weight of 65 kg, the figure Which is at in range the recommendation and reflects the efforts balance between meeting metabolic needs and prevention complications overfeeding on patients with disturbance ventilation heavy.

Election route nutrition on patient critical with failure breath need consideration careful, and second guide ESPEN and ASPEN/SCCM keep putting nutrition oral or enteral as the route of choice when the gastrointestinal tract is functioning well, given its benefits in maintaining the integrity of the intestinal mucosa and reducing the risk of infection compared to nutrition parenteral (Singer et al., 2023; Compher et al., 2022). However, on patient with distress heavy breathing Which Not yet intubated, giving nutrition Oral and enteral feedings are often hampered by extreme tachypnoea which increases the risk of aspiration, decreased consciousness, and nausea and vomiting due to hypoxemia and uraemia. On case this, it is depicted with clear transition order like nutrition follows changes in the patient's clinical status, starting from giving soft diet orally 2.100 kcal per day in the initial phase when the patient is still able to receive intake independently, then facing significant challenges in the form of decline intake due to persistent shortness of breath, nausea, and anorexia Which common found on patient with severe hypoxemia and uraemia secondary to acute kidney injury, a clinical picture which confirms that multiorgan failure in critical patients mutually burdens the ability to fulfil need nutrition in a way natural.

Acute kidney injury What happened during the course of this patient's illness added to the complexity of nutritional management because it required adjustments to the composition. macronutrients for minimize metabolic nitrogen load without excessively compromising the body's protein status. The AKI nutrition consensus recommends an energy requirement of 20–30 kcal/kgBW/day for all stages of AKI, with a protein intake of 0.8–1.0 g/kgBW/day in non-catabolic AKI patients who have not undergone renal replacement therapy, increased to 1.2–2.0 g/kgBW/day if there is an underlying disease that is catabolic, and just raised it higher (1.5–2.5 g/kgBW/day) if the patient is undergoing continuous renal replacement therapy due to

significant extracorporeal amino acid losses (Heyland et al., 2023; Hung et al., 2022; Otis et al., 2025). Restrictions protein on phase non-dialysis aimed at slowing down the accumulation of urea without triggering endogenous protein catabolism due to energy deficit, which can actually worsen azotaemia (Otis et al., 2025). In this case, the application of a non-dialysis kidney diet with protein 0.8 g/kgBW/day and energy 30 kcal/kgBW/day according to the recommendations of the nephrology division is consistent with the consensus range, especially since the patient and family consistently refused haemodialysis despite indications of AKI stage. heavy, so that Nutritional strategies are one of the main conservative pillars to slow down progressiveness disturbance kidney while still providing energy substrate for the lung healing process.

In addition to the quantitative aspects of calories and protein, nutritional support in ARDS also takes into account the qualitative composition of certain nutrients which theoretically can modulate the response. inflammation lungs, like fatty acid omega-3 chain long, gamma-linolenic ACID, and various antioxidants, which expected lower production of proinflammatory eicosanoid inflammatory mediators and improved oxygenation and lung mechanics. Several randomized clinical trials have shown improvements in secondary outcomes, such as ventilator-free days and oxygenation index with supplementation omega-3 in ARDS (Otis et al., 2025). However review systematic A Cochrane-based review previously reported that evidence regarding the benefits of immunonutrition formulas on overall mortality remains fragmented, inconsistent and quality low, so that its use outside the research context has not been recommended specifically routine by contemporary guides (Compher et al., 2022). In case This, therapy supportive addition in the form of N-acetylcysteine which has mucolytic properties as well antioxidants through donation group sulfhydryl for glutathione synthesis, given throughout treatment as part of oxidative stress mitigation strategies on lungs Which experience injury I on base fibrosis chronicle consequence pneumoconiosis.

Nutritional monitoring in critically ill patients with multiorgan dysfunction requires vigilance regarding metabolic complications like stress hyperglycaemia associated with worse clinical outcomes, electrolyte disturbances due to redistribution cellular and renal loss, as well as the risk of refeeding syndrome especially on patient with intake oral that had time decrease significant before optimized nutritional interventions. The 2023 ESPEN Guidelines emphasize that every patient critical Which treated more from four tens eight O'clock must considered at risk of malnutrition and requiring structured monitoring of metabolic parameters during nutrition administration (Fu et al., 2025). In this case, hyponatremia was found. And hypokalaemia light on initial evaluation is most likely related to a combination of inadequate oral intake, gastrointestinal losses through vomiting, and dysfunction kidney Which bother regulations normal electrolytes, so that monitoring electrolyte periodically and blood glucose checks every two hours twelve hours done during maintenance reflect clinical awareness of potential metabolic complications the.

Body position and ventilation strategy also interact closely. with success order like nutrition on ARDS, remembering elevation head bed is part of a standard care bundle to reduce the risk of aspiration and ventilator-associated pneumonia while optimizing mechanics breathing, while mobilization and periodic changes of position also play a role in prevention worsening skeletal muscle dysfunction accompanying prolonged immobilization on patient critical (Matthay et al., 2024). On case this, the application of thirty-degree head elevation and

changes position every two O'clock as part from the bundle maintenance intensive describe integrated efforts between respiratory and nutritional management Which each other support optimization patient clinical outcomes, although on Finally No can prevent development dysfunction diaphragm and failure weaning Which accompany ventilation invasive mechanics prolonged.

Limited diagnostic and therapeutic resources in regional health facilities also contribute to this important determinants in journey clinical features of this case, reflected in the unavailability of SARS-CoV-2 PCR testing for remove the differential diagnosis infection virus, as well as family's refusal of referral plans to more comprehensive health facilities or to other actions haemodialysis. Definition global ARDS The latest versions are explicitly designed to remain applicable on condition limitations source power, accommodate diagnosis-based saturation peripheral oxygen without the need for blood gas analysis or positive pressure ventilation devices (Fazzini et al., 2023; Grasselli et al., 2023). This condition confirms that the success of the management patient critical, including aspect nutrition, not solely dependent on accuracy protocol clinical, but also greatly influenced by systemic factors health, availability source Power, and the process decision-making together between medical team and family patient. Approach comprehensive involving intensivists, clinical nutritionists, and nephrologists in this case, although in the end it was not capable prevent worsening progressive to the patient die world, still represents optimal effort within the constraints of available resources, as well as give learning important regarding the complexity of nutritional management in ARDS secondary to lung disease consequence the work that accompanied by complications fail kidney.

CONCLUSION

Based on the findings of this study, it can be concluded that nutritional management in patients with acute respiratory distress syndrome (ARDS) secondary to pneumonoultramicroscopicsilicovolcanoconiosis represents a complex challenge influenced by the interaction of severe respiratory impairment, systemic inflammatory responses, renal dysfunction, and reduced ability to independently fulfil nutritional requirements. In this case, the hypermetabolic and hypercatabolic states caused by critical illness increased the patient’s energy and protein requirements; however, nutritional administration had to be adjusted according to the patient’s clinical progression, particularly after the development of acute kidney injury (AKI). Changes in the patient’s condition required continuous modification of the nutritional strategy, beginning with oral soft diet administration when the clinical condition permitted and subsequently transitioning to a non-dialysis renal diet with protein restriction according to recommendations after renal impairment occurred. Although multidisciplinary management involving respiratory support, nutritional intervention, and renal function monitoring was implemented, the disease continued to deteriorate, requiring invasive mechanical ventilation and ultimately resulting in death. This case demonstrates that nutritional therapy in ARDS patients with occupational lung disease and multiorgan complications should not focus solely on meeting calorie and protein requirements but also requires continuous evaluation of nutritional tolerance, organ function changes, potential metabolic complications, and overall clinical status. Future research is recommended to investigate larger patient populations through prospective observational studies or multicentre research designs to

provide stronger evidence regarding optimal nutritional strategies for ARDS patients associated with occupational dust-related lung diseases. Future studies may also evaluate the relationship between nutritional status, inflammatory biomarkers, ARDS severity, mechanical ventilation requirements, AKI incidence, and clinical outcomes more comprehensively. In addition, further research should explore the effectiveness of various nutritional approaches, including the timing of nutritional initiation, phase-specific energy and protein targets, and the application of specific nutritional interventions in patients with high catabolic risk. The development of evidence-based guidelines for patients with combined ARDS, occupational lung disease, and multiple organ dysfunction is necessary to provide healthcare professionals with more accurate decision-making frameworks when managing complex clinical conditions.

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