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Nutritional Support in Acute Respiratory Distress Syndrome (ARDS): A Comprehensive Literature Review On Clinical Strategies and Patient Outcomes

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Abstract

Introduction and purpose: Acute respiratory distress syndrome (ARDS) is a life-threatening condition and is a frequent reason for admitting patients to the Intensive Care Units. The treatment of ARDS remains an important task for physicians worldwide. Nutritional therapy is a significant part of managing critically ill patients. The aim of this study is to gather and present knowledge regarding nutrition support for patients diagnosed with ARDS.

A brief description of the state of knowledge: Acute Respiratory Distress Syndrome (ARDS) is a severe inflammatory lung condition associated with high morbidity and mortality. While respiratory support remains the cornerstone of treatment, nutritional therapy has emerged as a critical component of care. Patients with ARDS experience increased metabolic demands and catabolism, necessitating individualized nutritional strategies to preserve muscle mass, support immune function, and improve clinical outcomes. Although enteral nutrition is preferred,

parenteral routes are often required due to feeding intolerance or contraindications. Emerging evidence also supports the use of immune-modulating nutrients, such as omega-3 fatty acids and antioxidants, though clinical recommendations remain cautious pending further research. Refeeding syndrome remains a serious and potentially life-threatening complication necessitating careful monitoring and gradual reintroduction of nutrients.

Conclusions: Nutritional support is a vital component in the management of ARDS, with significant implications for patient recovery and outcomes. Individualized strategies that address energy and protein needs, minimize the risk of refeeding syndrome, and consider both enteral and parenteral routes are essential. While immune-modulating nutrition holds promise, further high-quality studies are needed to establish its routine use. Integrating nutrition into ARDS care pathways may enhance treatment effectiveness and improve prognosis.

Keywords: ARDS, nutritional therapy, parenteral nutrition

Introduction

Acute Respiratory Distress Syndrome (ARDS) presents a significant clinical challenge due to its complex pathophysiology, high mortality rate, and the absence of targeted pharmacological therapies. While advancements in ventilatory strategies and supportive care have improved outcomes, the role of nutrition has gained increasing recognition as a fundamental aspect of comprehensive management. Critically ill patients with ARDS often experience a hypermetabolic and catabolic state that contributes to rapid loss of lean body mass, impaired immune response, and prolonged mechanical ventilation. As such, appropriate nutritional support is not merely supportive, but also therapeutic. This article evaluates the current evidence on nutritional strategies in ARDS, including energy and protein requirements, the timing and route of nutrition delivery, complications such as refeeding syndrome, and the emerging role of immune-modulating nutrients. By examining these aspects, we aim to clarify the significance of individualized nutrition plans in improving clinical outcomes for patients with ARDS.

Materials and methods

A literature search was conducted using databases available in PubMed, Google Scholar and Elsevier with the following keywords: „nutrition in ARDS", „nutrition support in ARDS", „nutritional therapy in ARDS", „nutritional treatment in ARDS". We included only articles written in English. Additional references were identified using bibliographies of chosen articles. All retrieved studies were screened for relevance based on their titles, abstracts, and full texts. A total of 39 articles were used.

Description of the state of knowledge

Acute Respiratory Distress Syndrome: definition and diagnosis

The definition of ARDS was introduced for the first time in 1967. Since then, it was revised and changed several times. Nowadays, ARDS is diagnosed using the Berlin definition. It is described as an acute inflammatory lung injury and is characterized by the rapidly developing (within 1 week) state of clinically significant hypoxaemia with presence of diffuse pulmonary infiltrates that are visible in the chest imaging[1]. Additionally, respiratory failure cannot be fully explained by heart failure or fluid overload. Criteria for diagnosing ARDS include a partial pressure of oxygen to fraction of inspired oxygen ratio (PaO_2:FiO_2 ratio) of 300 mmHg or less measured on at least 5 cm H_2O PEEP or CPAP (continuous positive airway pressure). Oxygenation can also be used for determining the severity level of ARDS: mild- 200 mmHg < PaO_2:FiO_2 ≤ 300 mmHg; moderate- 100 mmHg < PaO_2:FiO_2 ≤ 200 mmHg;
severe- PaO_2:FiO_2 ≤ 100 mmHg.

Imaging is an important part of diagnosing ARDS. Although a chest X-ray is usually sufficient, a CT scan may provide additional data about the patient's condition and an underlying cause. Furthermore, contrast-enhanced CT imaging may detect concurrent pulmonary embolism, a finding that could significantly influence therapeutic management. Recent advancements in ARDS diagnosis include the integration of point-of-care ultrasound (POCUS) by bedside clinicians, combining lung ultrasound and focused cardiac echocardiography[2]. Lung ultrasound facilitates ARDS detection by identifying interstitial syndromes such as inhomogeneous, gravity-independent B-lines, pleural line thickening with reduced lung sliding, spared lung areas, and subpleural consolidations. Focused cardiac ultrasound contributes to the diagnostic process by helping to exclude cardiogenic pulmonary edema and assessing right ventricular function. Right ventricular dysfunction may manifest as acute cor pulmonale, characterized by right ventricular dilation and septal dyskinesia.

Acute Respiratory Distress Syndrome: causes

The causes of ARDS are varied and multifactorial. It can be caused by both infectious and non-infectious triggers with the pulmonary sepsis (ie. pneumonia) being the most common cause. Other causes include non-pulmonary sepsis, pancreatitis, aspiration of gastric contents, severe traumatic injuries with shock and multiple transfusions. Recently, a new appearing trigger of ARDS is e-cigarette and vaping-associated lung injury. In the last few years, public awareness of ARDS has increased due to the COVID-19 pandemic, during which SARS-CoV-2 has been the leading cause of respiratory failure.

Acute Respiratory Distress Syndrome: pathology and pathophysiology

The pathophysiology of ARDS is complex and not fully understood. It varies depending on the causative factor and individual predisposition. The mechanism of ARDS involves the activation and disruption of several injury response pathways, along with inflammation and coagulation processes occurring both in the lungs and systemically. Although these responses are integral to the normal host defense against infection or injury, their excessive activation can lead to tissue damage. First, neutrophils infiltrate the alveolar space and release injurious mediators such as reactive oxygen species, proteases, and proinflammatory lipid-derived mediators such as prostaglandins and leukotrienes. They also form extracellular traps that activate the NLRP3 inflammasome, promoting cytokine release and amplifying inflammation. Neutrophil recruitment is primarily mediated by macrophages and epithelial cells via cytokines and chemoattractants like Interleukin-8. Migration occurs mainly through paracellular pathways, possibly regulated by fibroblasts. In addition to neutrophils and macrophages, other immune cells, including lymphocytes and dendritic cells, contribute to inflammatory regulation. Systemic inflammation is common and likely contributes to extrapulmonary organ dysfunction, particularly affecting the kidneys and brain—both associated with worse ARDS outcomes[3].

The hallmark histopathological feature of ARDS found in some of autopsy cases is diffuse alveolar damage (DAD). DAD is characterized by neutrophilic alveolitis and the presence of hyaline membranes. Additional pathological findings include bilateral pneumonia and, less frequently, diffuse alveolar hemorrhage. DAD reflects significant injury to both the alveolar epithelium and pulmonary endothelium, particularly affecting the integrity of the alveolar–capillary barrier, a central element in ARDS pathogenesis.

Acute Respiratory Distress Syndrome: management

Most patients with ARDS are treated in the Intensive Care Units. A vital element of treatment is proper oxygen therapy. Lung protective ventilation requires the use of low tidal volume and

targeting low airway pressures[4]. Prone positioning is deemed to be beneficial and to reduce mortality[5]. Milder cases of ARDS can sometimes be managed with non-invasive respiratory support such as HFNO (High-Flow Nasal Oxygen). In the most critical cases of ARDS, veno-venous extracorporeal membrane oxygenation (ECMO) may be employed as a life-saving intervention.

Currently, there is no universal pharmacological therapy for the treatment of ARDS[6]. Initially, the focus should be on treating the reversible causes of the disease. This is especially important if the cause is infectious, when antibiotics or antiviral drugs become of great importance. Some studies show the benefit of using systemic corticosteroids although the results are inconclusive. Nutritional support is a fundamental aspect of care in critically ill patients. It has progressed beyond simply supplying energy and essential nutrients in proper ratios to taking advantage of the pharmacological properties of carbohydrates, amino acids, and lipids. ARDS is associated with a heightened proinflammatory state and increased catabolic activity, which can result in substantial nutritional deficiencies. Providing nutritional support is therefore essential to avoid cumulative energy deficits, malnutrition, loss of lean muscle mass, and decline in respiratory muscle function.

Route of nutrition: enteral vs parenteral

Most patients with ARDS are ventilated, which prevents them from receiving nutrition orally. Those patients can receive nutrition in two ways- either enteral (EN) or parenteral (PN). Enteral route includes nasogastric or nasojejunal tubes for short-term access and gastrostomy, jejunostomy or gastrojejunostomy tubes for long-term nutrition[7,8]. While gastric access should be chosen for most patients, postpyloric tube placement is recommended for those with high risk of aspiration, gastric feeding intolerance not solved with prokinetic agents or gastroparesis. Parenteral nutrition is delivered intravenously with appropriate catheter. Access for PN can be obtained using simple IV line (for temporary use only), peripherally inserted central catheter (PICC) line or central catheter. If the expected duration of PN is more than a month, a tunneled central venous catheter (e.g. Broviac) should be taken into consideration. While enteral nutrition is considered superior to parenteral nutrition due to being more physiological, natural and less risky, it cannot be used in all patients. There are numerous indications for administering PN, either exclusive or supplemental (see Table 1)[9]. Additionally, the need for prone positioning in ARDS patients necessitate careful consideration of feeding strategies. When enteral nutrition is not well-tolerated, does not cover energy needs or is not fully digested, supplemental parenteral nutrition added to a reduced volume of EN can be a beneficial

therapeutic option[10,11]. Exclusive parenteral nutrition should be reserved for absolute contraindication to enteral nutrition and should not be implemented until all reasonable methods of improving EN tolerance have been tried. However, parenteral nutrition is preferable to delayed enteral nutrition and should be administered when required[12].

Table 1. Indications for parenteral nutrition

- Enteral nutrition intolerance- persistent diarrhea or vomiting
- Prolonged ileus
  - Mechanical obstruction
  - Generalized peritonitis
  - Peritoneal carcinosis
  - Abdominal distension on EN
- Short Bowel syndrome
  - Mesenteric infarction
  - Extensive small bowel resection
- Unrepaired anastomotic leak
- Severe malabsorption
  - Radiation injury to intestine
  - High output fistula (internal or external)
  - Inflammatory bowel diseases in acute phase
  - Splanchnic ischemia
- Time to meet full energy needs with EN >5 days
- Insufficient energy or nutrients intake
- Inability to gain or maintain weight
- High risk of aspiration

Determining energy and nutrient requirements

Determining energy requirements is crucial for administering adequate nutrition. Both overfeeding and underfeeding can negatively affect the patient. Nutritional management in ARDS patients is complex due to the hypermetabolic and hypercatabolic nature of the syndrome, often compounded by comorbid conditions that significantly alter metabolic demands. Standard predictive equations (e.g., Harris-Benedict or weight-based formulas) often fail to capture the dynamic shifts in energy expenditure observed in critical illness and are associated with high inaccuracy[13]. As a result, indirect calorimetry (IC) is recommended as the

gold standard for measuring resting energy expenditure (REE), allowing for individualized and adaptive nutritional strategies. However, practical limitations such as cost, equipment availability, and technical factors (e.g., high FiO_2 or ventilator settings) often limit its use[14]. When IC is unavailable, guidelines support the use of ventilator-derived CO_2 production (VCO_2) as a more accurate alternative to predictive equations[15]. During the early phase of ARDS, hypocaloric feeding (not exceeding 70% of estimated energy needs) is advised to avoid overfeeding, which can exacerbate hypercapnia, increase infection risk, and delay ventilator weaning[16]. After the acute phase (typically after the third day), energy intake can be gradually increased to 80–100% of the measured or estimated REE to support recovery while avoiding cumulative energy deficits. Hypocaloric feeding should not be maintained for a prolonged period as it can lead to malnutrition, loss of lean body mass, and reduced respiratory muscle strength which are associated with poor outcomes, including prolonged mechanical ventilation and increased mortality[17–19].

Protein provision is equally critical in ARDS, as protein-energy malnutrition leads to the loss of skeletal muscle, including respiratory musculature. Guidelines recommend a progressive increase in protein intake, aiming for up to 1.3 g/kg/day of protein equivalents as tolerated[20]. The composition of macronutrients also requires consideration. Earlier concerns that high-carbohydrate diets could worsen hypercapnia have been largely refuted; it is now understood that total caloric excess, rather than carbohydrate proportion alone, more closely correlates with increased carbon dioxide production. Therefore, balanced macronutrient delivery tailored to individual tolerance and metabolic status remains essential. Excessive fat intake may also result in hepatic steatosis and immunosuppression, emphasizing the need for careful monitoring.

Immune-modulating nutrition

In acute respiratory distress syndrome (ARDS), characterized by an overwhelming inflammatory response and disruption of the alveolar-capillary barrier, immune-modulating nutrition has emerged as a supportive strategy aimed at mitigating pulmonary inflammation and improving clinical outcomes[21–36]. Specialized enteral formulas enriched with omega-3 polyunsaturated fatty acids (n-3 PUFAs)—particularly eicosapentaenoic acid (EPA), docosahexaenoic acid (DHA), γ-linolenic acid (GLA), and antioxidants—exert their effects through multiple immunological and biochemical mechanisms. These nutrients modulate inflammatory pathways by displacing arachidonic acid in cell membranes, thereby altering eicosanoid synthesis toward the production of less pro-inflammatory and more anti-inflammatory lipid mediators, such as resolvins and protectins. EPA and GLA supplementation

increases the formation of dihomo-γ-linolenic acid (DGLA) and reduces leukotriene B4 and prostaglandin E2, leading to decreased neutrophil activation, endothelial injury, and alveolar permeability. Antioxidants such as vitamin E and β-carotene mitigate oxidative stress by scavenging reactive oxygen species, further protecting lung tissue from free radical-induced damage.

Clinically, these mechanisms translate into improved oxygenation (as reflected in higher PaO2/FiO2 ratios), reduced alveolar inflammation, and enhanced lung compliance. Studies have shown that continuous administration of EPA/GLA-enriched formulas leads to significant reductions in ventilator days, ICU stay, and even mortality in certain patient subsets. However, the benefits appear less pronounced when administered as a bolus or in combination with other immunonutrients such as arginine, which may have deleterious effects in critically ill patients. Despite promising biological plausibility and supportive early trial data, clinical recommendations remain guarded due to variability in study designs, formulations, and inconsistent outcome measures across trials.

Refeeding syndrome

Refeeding syndrome (RFS) is a possible complication of nutritional therapy in critically ill patients in the ICU. RFS is a result of reintroduction of calories after a time of undernourishment. It is probable with any type of feeding- oral, enteral or parenteral with the greatest risk associated with enteral nutrition. RFS can also develop when the beginning of nutritional therapy is delayed by more than 5 days, especially in patients who are metabolically stressed due to severe illness, including patients with ARDS. Currently, there is no standardized definition of RFS. Most scientific societies agree that RFS can be described as falls in serum levels of phosphate, potassium and/or magnesium within 5 days of reintroduction of previously significantly decreased caloric intake[37]. Thiamin (vit. B1) deficiency may also follow mentioned electrolytes imbalance. As RFS is potentially lethal, implementing preventive methods is crucial. The first step in prevention involves identifying patients at particularly high risk of developing RFS (see Table 2). After identifying those patients, additional precautions should be taken, such as slow initiation and advancement of nourishment, measuring electrolyte levels at least once daily for the first week, electrolyte supplementation and thiamine administration. In patients with refeeding hypophosphatemia, energy supply should be restricted for 48 hours, then gradually increased and electrolytes should be measured two to three times per day and supplemented if needed[38,39].

Table 2. Refeeding syndrome risk factors

- Starvation and malnourishment
  - BMI <18,5
  - 5% weight loss in 1 month
  - Loss of subcutaneous fat and muscle mass
- Polymorbidity and polypharmacy
- Elderly
- Low serum magnesium (<0,7 mmol/L)
- Malabsorption
  - Short bowel syndrome
  - Bariatric surgery
  - Inflammatory bowel diseases
  - Radiation enteritis
  - Cystic fibrosis
  - Pancreatic insufficiency
- Hemodialysis
- Chemotherapy
- Addictions (e.g. alcohol, drugs)
- Anorexia nervosa

Conclusions

Acute Respiratory Distress Syndrome (ARDS) is a severe inflammatory lung condition with diverse causes, primarily sepsis and pneumonia. Diagnosis relies on the Berlin definition, supported by imaging and bedside ultrasound. While management includes mechanical ventilation, prone positioning, and supportive care, nutrition plays a critical role in improving outcomes. ARDS induces a hypermetabolic, catabolic state requiring individualized nutritional support. Enteral nutrition is preferred, though parenteral nutrition is used when enteral feeding is insufficient or contraindicated. Protein delivery up to 1.3 g/kg/day is essential to preserve muscle mass. Indirect calorimetry is recommended to guide energy needs. Refeeding syndrome must be actively prevented in high-risk patients. Immune-modulating nutrition with omega-3 fatty acids and antioxidants may reduce inflammation and improve oxygenation, although further evidence is needed to support its routine use.

Disclosure:

Authors' contribution

Conceptualization: MJ and KS;

Methodology: MJ and NK;

Software: KS and JMM;

Check: AD, KS and JMM;

Formal analysis: JMM;

Investigation: MJ and KS;

Resources: NK and AD;

Data curation: JMM;

Writing- rough preparation: MJ and KS;

Writing - review and editing: KS, JMM and AD;

Visualization: AD and NK;

Supervision: KS;

Project administration: MJ;

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