JOURNAL OF NUTRITION COLLEGE
Volume 14, Nomor 3, Tahun 2025, Halaman 276-281
Received: 1 Januari 2025, Revised: 16 Februari 2025
Accepted: 17 Februari 2025, Published:30 Juli 2025
Online di: http://ejournal3.undip.ac.id/index.php/jnc/ , DOI:10.14710/jnc.v14i3.48773

COMPREHENSIVE NUTRITIONAL THERAPY ON A 60-YEAR-OLD MALNOURISHED MALE PATIENT WITH PULMONARY ADENOCARCINOMA AND ESOPHAGEAL STENOSIS

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ABSTRACT

Background: *Pulmonary adenocarcinoma with esophageal stenosis can trigger severe malnutrition due to catabolism, inflammation, and anabolic resistance compounded by impaired oral intake.*
Objective: *To Describe the nutritional challenges and impact enteral nutrition strategy used, evaluate its impact on body weight, nutritional status and functional capacity. Evidence based nutritional support*
Method: *The Subject was selected based on unique clinical feature. Malnourished patient's journey with pulmonary adenocarcinoma and esophageal stenosis enteral nutrition via a gastrostomy tube. 60-year-old male with Shortness of breath, cough and throat obstruction causing vomiting and difficulty swallowing. Biomarker complete blood count, electrolyte test, body weight changes, BMI, Barthel index scale. Weight loss exceeding 10kg within three months, required assistance for daily activities. Examination severe loss of subcutaneous fat and muscle mass, Bedridden status. Body weight of 54kg, height 172cm, BMI of 18,25kg/m². Laboratory indicated anemia and hyponatremia RBC (3.70 x 10^6/µL), HGB (7.8 g/dL), HCT (26.2%), MCV (70.8fL), MCH (21.1pg), MCHC (29.8g/dL), and Na (121mmol/L). Diagnosed in January 2024 with pulmonary adenocarcinoma with esophageal stenosis, necessitating gastrotomy tube. Nutritional intervention adjustments from oral to enteral feeding formulas and caloric targets. Gradual increase nutritional therapy, initial target of 40kcal/kg and 1.3g protein/kg, providing 2160kcal and 85g protein daily. The diet began at 15 kcal/kg, with supplementation. Follow-ups reaching 2560kcal and 100g protein. Assessment since July 6 until October 14. Location at Bali Mandara Hospital.*
Results: *Weight gain 54 kg to 61 kg. an increase in caloric intake to 2500 kcal/day, tolerated enteral feeding well, without complication. Improved energy, improve functional capacity, better lab values, preparing him for radiotherapy. Caloric escalation countering anabolic resistance. Protein intake stimulates muscle synthesis. Omega-3fattyacids reduce inflammation, Zinc support immunity, Vitamin B6 support hematopoiesis, VCO quick energy for hypermetabolism.*
Conclusion: *Comprehensive nutritional therapy with supplementation improved nutritional status and stabilized metabolic state in this patient.*

Keywords *: malnutrition; pulmonary adenocarcinoma; esophageal stenosis; nutritional therapy*

INTRODUCTION

Pulmonary adenocarcinoma is a non-small cell lung cancer that originates in the glandular cells of the lung's epithelial tissue, one of the leading causes of cancer-related mortality worldwide. Originates in the peripheral airways and alveolar regions of the lung, characterized by glandular differentiation and mucin production. Pulmonary adenocarcinoma is a complex malignancy by genetic mutations and alterations cellular signaling pathways. the malignant transformation of epithelial cells in the distal lung, typically affecting the alveolar cells. Molecular changes in pulmonary adenocarcinoma include mutations in genes such as *EGFR* (epidermal growth factor receptor), *KRAS* (Kirsten rat sarcoma viral oncogene homolog), *ALK* (anaplastic lymphoma kinase), and *ROS1*, which contribute to uncontrolled cellular proliferation and resistance to apoptosis.[1] The mutation of *EGFR* is particularly notable as it is associated with better responses to targeted therapies with tyrosine kinase inhibitors (TKIs).[2] the expression of the tumor suppressor gene *TP53* mutations, which are present in approximately 50% of cases, contributing to genomic instability. Other pathways, such as the PI3K/AKT/mTOR pathway, also play a role in disease progression.[3] The tumor microenvironment (TME), which includes stromal cells, immune cells, and extracellular matrix components, further supports tumor growth and metastasis through

various molecular interactions.[4] The primary risk factors for pulmonary adenocarcinoma mirror those for other forms of lung cancer. Tobacco smoking is the leading cause of lung cancer, although adenocarcinoma is more commonly associated with non-smokers compared to other subtypes of lung cancer. This reflects the distinct genetic and molecular characteristics of adenocarcinoma, with a higher prevalence of mutations in non-smokers and lighter smokers, particularly those involving *EGFR* and *ALK*.[5,6]

Other risk factors include exposure to second-hand smoke, environmental toxins (such as radon, asbestos, and air pollution), and a family history of lung cancer. Genetic predispositions, such as inherited mutations in certain tumor suppressor genes, can also contribute to increased susceptibility.[7] Pulmonary adenocarcinoma often presents with nonspecific symptoms, which can delay diagnosis. Common clinical manifestations include a persistent cough, dyspnea (shortness of breath), and hemoptysis (coughing up blood), which are generally more subtle compared to other lung cancer subtypes.[8] Due to its peripheral origin, adenocarcinomas tend to remain asymptomatic until the tumor has reached a larger size or has metastasized to other organs, such as the brain, liver, or bones.

Symptoms of metastasis can include weight loss, fatigue, chest pain, and neurological symptoms, depending on the site of spread. In advanced stages, patients may experience pleuritic pain and symptoms of pleural effusion, as the tumor invades the pleura.[9] Imaging studies such as chest X-rays or CT scans are typically used to detect lesions, and biopsies confirm the diagnosis.[10]

Esophageal stenosis is a narrowing of the esophagus leading to difficulty swallowing (dysphagia) and potential obstruction. It can be caused by a variety of conditions, ranging from chronic inflammation and scarring to malignancies. both squamous cell carcinoma and adenocarcinoma, can lead to esophageal stenosis as the tumor invades the esophageal wall, leading to a narrowing of the esophageal lumen. In particular, esophageal adenocarcinoma, commonly associated with Barrett's esophagus and chronic GERD, can cause significant stenosis.[11] Tumors can develop in the middle or distal portions of the esophagus, where they often present as strictures. The coexistence of Pulmonary adenocarcinoma with esophageal stenosis is rare but significant case leads to severe malnutrition from excess catabolism, chronic inflammation, anabolic resistance aggravated by inability taking oral nutrition. This case report highlights the impact of comprehensive nutritional therapy on a malnourished patient with these conditions who has not undergone chemotherapy or radiotherapy. *Unlike standard protocols that follow fixed nutritional targets based on patient's complex case, our intervention involved a progressive increase in caloric intake from 2360 kcal to 2560 kcal, guided by weight trends and patient's feeding tolerance. The challenge of maintaining nutritional stability in cancer-related inflammation and suggests that early, adaptive feeding strategies may improve outcomes in similar patients.*

METHOD

The subject for this case report was selected based on clinical significance and educational value for nutritional management in complex case of oncology patient. Data for this case report were collected through a review of the patient's medical records, direct interviews, clinical examination assessment, and nutritional monitoring. The data collection process included: Anthropometric measurements body weight trends recorded, Body Mass Index calculated trends, change in nutritional status following the nutritional intervention progression, Laboratory data, functional status improvements. This case report was prepared with patient consent, confidentiality maintained. Patient history A 60-year-old male presented with worsening shortness of breath, cough, fatigue, chest pain, dysphagia, and throat pain over the past month. He reported appetite loss, inadequate food intake, and over 10 kg weight loss in three months, leading to weakness and dependency for daily activities. He experienced throat obstruction, frequent post-meal vomiting, and worsening swallowing difficulties. Anthropometry Examination revealed a weight of 54 kg, height of 172 cm, BMI of 18.25 kg/m², extensive fat and muscle loss, and bedridden status. Nutrition screening using Subjective Global Assessment shows score C more than 50% with significant physical signs indicates nutritional diagnosis of severe malnutrition based on unintentional body weight loss more than 10% in less than 3 months, there's food intake disturbance with inadequate food intake and severely decreased compared to before illness, Degree of dietary intake change: unable to eat with drastic changes. with gastrointestinal symptoms, difficulty eating due to vomiting everytime drinking or eating, Decline in functional capacity, bedridden with no physical activity, Severely increased metabolic stress due to malignancy and extensive fat and muscle loss. Biochemical markers Blood parameters showed anemia (RBC 3.70 x 10^6/µL, hemoglobin 7.8 g/dL)

and hyponatremia (sodium 121 mmol/L). Diagnosed with pulmonary adenocarcinoma in January 2024, further evaluation revealed he had esophageal stenosis requiring gastrostomy tube placement. From the moment patient is diagnosed, he declined both chemotherapy and radiotherapy. Nutritional therapy became the cornerstone management plan.

Dietary Intake Nutritional Intervention plan targeted 40 kcal/kg and 1.3 g protein/kg of ideal body weight (2160 kcal, 85 g protein/day). The diet began at 15 kcal/kg, supplemented with virgin coconut oil (10 ml every 4 hours), zinc (20 mg every 12 hours), omega-3 (1 g daily), and vitamin B6 (10 mg every 8 hours). After hospitalized for 10 days, the patient had follow-ups every three weeks at clinical nutrition clinic, during which caloric intake was increased by 200 kcal increments and protein adjusted to 1.5 g/kg of ideal body weight, eventually reaching 2360–2560 kcal and 100 g protein/day. The calorie and protein requirements are subsequently met with oral nutrition supplements, the addition of VCO (virgin coconut oil), juice, egg whites, and blended meat.

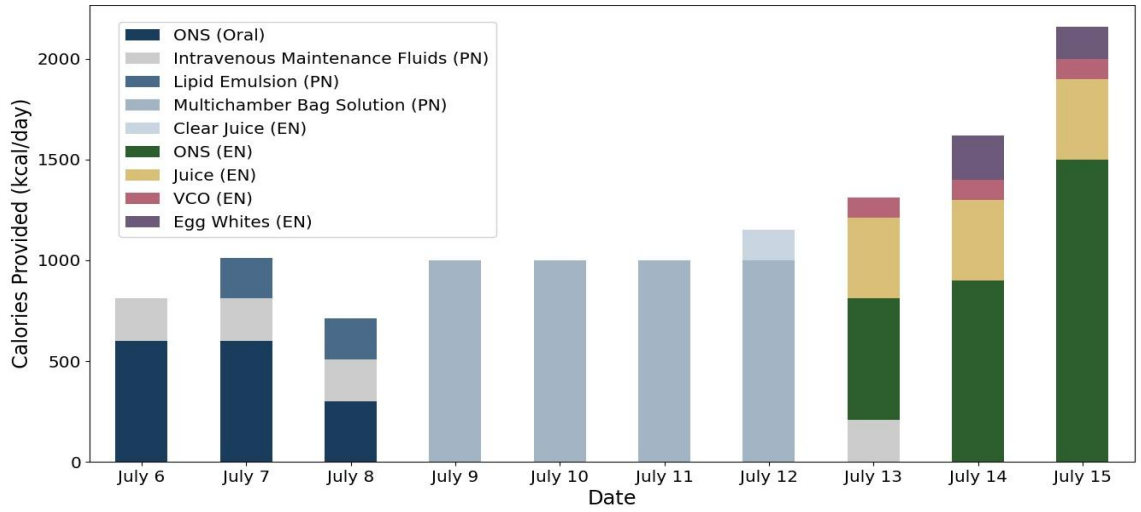

Figure 1. Daily progress caloric intake by nutrition type and method during 10-days hospitalization, The patient transitioned from oral and intravenous intake to intravenous only in preparation for surgery. After surgery, transitioned to combination of enteral and intravenous nutrition, and finally, the patient was able to meet the initial target caloric intake through the gastrostomy tube.

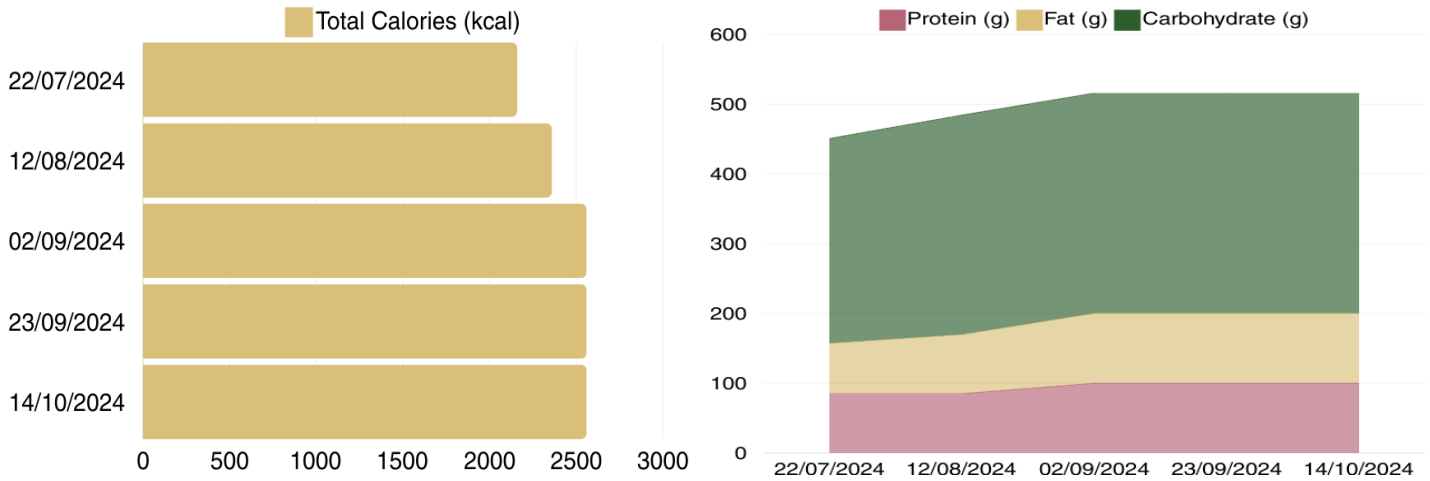

Figure 2 and 3. Bar chart and Area chart of total caloric and macronutient outpatient follow-ups. Represents the monitoring progression of increased caloric intake and transition in macronutrient composition fat, protein, and carbohydrates during follow-up visits at the nutrition clinic.

RESULTS

Blood parameters improvements after 10 days post hospitalization: RBC (3.70 to 4.52 x 10^6/µL), HGB (7.8 to 10 g/dL), HCT (26.2% to 33%), MCV (70.8 to 73 fL), MCH (21.1 to 22.1 pg), MCHC (29.8 to 30.3 g/dL), and Na (121 to 126 mmol/L). After being discharged from the hospital, the patient attended regular follow-up appointments at the clinical nutrition clinic, and these findings were identified from routine outpatient monitoring every three weeks and the patient being observed for four months. Based on the physical examination during the most recent outpatient clinic visit, the patient's nutritional status has improved from severe malnutrition to good nutrition. With the support of their family, the patient is now able to achieve adequate nutritional intake through the gastrostomy tube without any difficulties or complications, Weight gain: Increased from 54 kg to 61 kg. BMI improvement: From 18.25 kg/m² to 20.61 kg/m². Functional capacity improved from severe to moderate dependency, increased independence in daily activities, better nutritional status, and enhanced readiness for radiotherapy. (bartle index score from 35 at first treatment and 80 after 4 months of nutritional therapy).

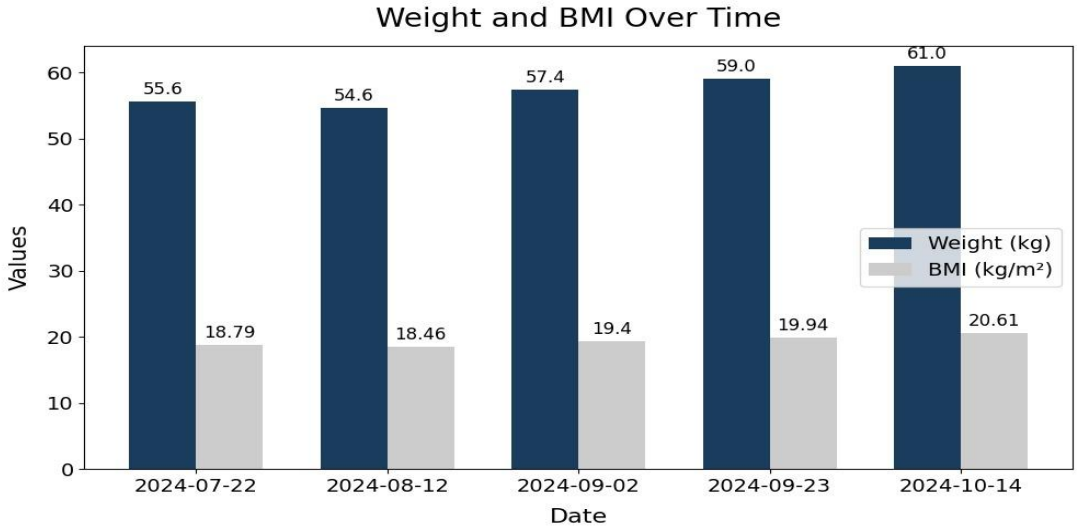

Figure 4. Bar chart. Weight And BMI Progress Over Time

DISCUSSION

Malnutrition in pulmonary adenocarcinoma with esophageal stenosis is multifactorial, arising from the tumor's metabolic demands and inflammatory responses. The Warburg effect aerobic glycolysis by cancer cells leads to systemic glucose depletion and energy imbalance. Elevated pro-inflammatory cytokines (IL-1, IL-6, TNF-α) activate ubiquitin-proteasome pathway, inducing proteolysis, muscle wasting, and compromised immunity. [2,3] These catabolic processes exacerbate malnutrition and diminish functional capacity. In this case, comprehensive nutritional therapy was implemented to counteract metabolic disruptions, support tissue repair and function. Resulted significant improvements in the patient's nutritional status, hematologic parameters, and overall functionality.[12,13]

The decision to implement enteral nutrition via a gastrostomy tube was based on ESPEN guidelines, which recommend enteral feeding preferred route for patients with dysphagia due to esophageal stenosis to prevent malnutrition, to achieve caloric target, emphasize early and individualized interventions. Patients with malignancies typically 25-30 kcal/kg/day and protein needs 1.2-1,5 g/kg/day. nutritional needs are elevated 40 kcal/kg/day due to patient's malnourished condition caused by hypermetabolism and inflammation. Because before admission patient have difficulty of swallowing and make the patient have minimum to zero intake and then to prevent refeeding syndrome, caloric intake was initiated at 15 kcal/kg/day and incrementally increased by 20–25% daily to reach 40 kcal/kg/day (2160 kcal) within 7–10 days. Outpatient follow-ups gradual increases of total caloric of 200kcal (2360-2560 kcal). Gradual approach avoided rapid insulin surges that cause life-threatening electrolyte shifts, such as hyponatremia and hypophosphatemia. Protein intake optimized to 1.3–1.5 g/kg of ideal body weight (85–100 g/day), stimulating the mTOR (mammalian target of rapamycin) pathway to activate anabolic processes essential for muscle repair and growth, High-protein intake was administered due to the patient's initial bedridden status, which is indicative of severe muscle weakness. Given that cancer-related malnutrition and inactivity contribute to muscle

atrophy, an increased protein supply was provided to support muscle regeneration and prevent further loss of lean body mass. Over time, this nutritional intervention contributed to improved functional capacity, allowing the patient to regain some independence in daily activities and transition from a bedridden state to partial mobility. High energy protein ONS and soft blended foods provided essential amino acids, compensating for potential malabsorption due to gastrostomy tube feeding route. the administration of omega-3 fatty acids was shown to help reduce inflammation, thereby enhancing the body's ability to utilize nutritional therapy more effectively. Omega-3s have been evidenced to modulate inflammatory responses in cancer patients, This was reflected in the gradual increase in the patient's body weight over time, indicating improved nutritional status and metabolic efficiency. Omega-3 fatty acids, EPA and DHA reduce systemic inflammation by modulating COX and LOX pathways, lead to production of anti-inflammatory prostaglandins and leukotrienes, enhance muscle protein synthesis via the Akt-mTOR pathway .[12,13]

This nutritional therapy led from 54 kg to 61 kg the percentage 12.96% increase body weight without adverse effects, improvements muscle strength, and enhanced functional capacity. The nutritional diagnose from malnourished to well nourished patient. Controlled caloric increases for metabolic adaptation, preventing cardiac and respiratory complications while promoting anabolic processes. Supplementation with zinc and vitamin B6 given to the patient because patient have wound scars after gastrotomy tube placement and also because of the anemia condition. Both supplementation supported immune function, wound healing, and hematologic recovery, evidenced by increase HGB levels to 10 g/dL and no inflammation or infection reported on patient's wound scar. Medium-chain triglycerides from VCO is given to the patient as the caloric addition and easily absorbable energy source requiring minimal oxygen for metabolism so will not burden patient's pulmonary malignancy condition, reducing metabolic burden on compromised respiratory function. This case demonstrates individualized nutritional therapy is effective in managing cancer-related malnutrition and significantly enhances patient outcomes.[14,15,16]

CONCLUSIONS

Comprehensive nutritional therapy, supplemented with individualized interventions, significantly improved the patient's nutritional status and stabilized their metabolic state. This approach effectively addressed severe malnutrition, reduced the risk of complications, and enhanced the patient's readiness for curative radiotherapy. Furthermore, optimizing nutritional support in this manner plays a critical role in improving outcomes from subsequent chemotherapy or radiotherapy, particularly in malnourished patients with pulmonary adenocarcinoma and esophageal stenosis.

ACKNOWLEDGEMENT

I would like to thank dr. Dwi Asti Lestari, Sp.GK for her guidance, mentorship and support throughout this case report. I also appreciate the healthcare team for their help in patient care and data collection. Finally, I am grateful to my family who have always supported me by providing a place to stay and good food, allowing me to think clearly.

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