Rehabilitation Nutrition for Acute Heart Failure on Inotropes with Malnutrition, Sarcopenia, and Cachexia: A Case Report

MALNUTRITION COMMONLY occurs in patients with heart failure. In patients with acute heart failure, 23.1% are malnourished and 51.9% are at risk of malnutrition.[1] Malnourished patients with heart failure can experience all causes of adult malnutrition, such as acute illness or injury, chronic illness, and social or environmental circumstances.[2] An association has been demonstrated between heart failure and micronutrient status, such as deficiencies in vitamins A, C, D, and E, thiamine, other B vitamins, selenium, zinc, and copper.[3] Cachexia, a complex metabolic syndrome associated with underlying illness and characterized by muscle loss with or without a loss of fat mass,[4] significantly decreases quality of life and survival in patients with heart failure.[5] Active screening for malnutrition and individualized dietary interventions may be appropriate in patients with cardiac cachexia.[5] Exercise-based rehabilitation for patients with chronic heart failure confers important improvements in exercise capacity and health-related quality of life, and reduces the risk of hospital admissions.[6] Aerobic exercise training for cardiac cachexia can also recycle and enhance protein expression and enzyme activities, thereby counteracting impairments in metabolism and muscle atrophy.[7] Therefore, nutritional management and rehabilitation are both imperative in patients with heart failure.

Sarcopenia can be common in patients with heart failure. Sarcopenia is a syndrome characterized by progressive and generalized loss of skeletal muscle mass and strength.[8] Primary sarcopenia is age-related when no other cause is evident. Secondary sarcopenia is considered when one or more other causes are evident, such as inactivity, disease, and/or malnutrition.[8] Muscle wasting has been detected in 19.5% patients with chronic heart failure.[9] Therefore, combating sarcopenia in patients with heart failure is important.

Rehabilitation nutrition is a combination of both rehabilitation and nutrition care management with the purpose of maximizing functionality in people with disabilities.[10] Rehabilitation nutrition may improve outcomes in malnourished and disabled patients with heart failure. However, no case of heart failure with malnutrition, sarcopenia, and cachexia treated by rehabilitation nutrition has been reported. In this article, we report a case of rehabilitation nutrition for heart failure complicated with malnutrition, sarcopenia, and cachexia.

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PATIENT PROFILE

The patient is a 39-year-old man who was admitted to the hospital immediately due to dyspnea at rest. He had a 1-month history of exertional dyspnea and was diagnosed with acute heart failure and suspected dilated cardiomyopathy. At the time of admission, he had atrial tachycardia with a heart rate of 160 beats per minute. The patient's height (182 cm), body weight (84.5 kg), and body mass index (BMI [calculated as kg/m^2] 25.2) were documented. He had gained 3 kg of body weight during the 10 days before admission. Echocardiography revealed that his left ventricular ejection fraction was 10%, his left ventricular end-diastolic diameter was 70 mm, and severe mitral regurgitation had developed. Dobutamine and milrinone were initiated to treat severe low cardiac output syndrome and improve inadequate end-organ perfusion. Intravenous amiodarone and repeated catheter ablation were used to treat refractory atrial tachycardia. Because his case was complicated by repeated septic shock caused by methicillin-sensitive *Staphylococcus aureus* and *Serratia marcescens*, he needed a longer intensive care unit stay.

The ethics committee of the Yokohama City University Medical Center approved the case study. The patient agreed to the publication of his case by providing informed consent. The investigations and case study were performed in accordance with the ethical standards established in the 1964 Declaration of Helsinki and later amendments, and ethical guidelines for medical and health research involving human subjects in Japan.

ASSESSMENT AND TREATMENT

Thiamin and iron concentrations at admission were 20.3 ng/mL (60.09 nmol/L) and 35 μg/dL (6.26 μmol/L), respectively, and thus thiamin and iron were supplemented during hospitalization. Total parenteral nutrition was started on day 3 because the patient was demonstrating hemodynamic instability. Parenteral administration of the inotropes was necessary to treat his hemodynamic instability. Supportive psychotherapy, including psychological counseling and drug therapy, was started from day 9. Zinc and copper concentrations at day 10 were 70 μg/dL (10.7 μmol/L) and 199 μg/dL (31.24 μmol/L), respectively. Enteral nutrition was initiated on day 14 and physical therapy was prescribed on day 22. He was referred to the nutrition support team on day 43 and leaving the cardiac care unit on day 47. The nutrition support team consisted of doctors, registered dietitian

nutritionists, physical therapists, nurses, pharmacists, and clinical laboratory technologists, who were all involved with the patient until hospital discharge. The nutrition support team intervened with the patient's treatment only after a referral from the attending physician.

Upon referral to the nutrition support team, the patient's body weight was 65.1 kg and his BMI was 19.7. His arm circumference was 23.5 cm and his triceps skinfold was 11 mm. Laboratory tests revealed the following: serum albumin 2.7 g/dL (27 g/L); hemoglobin 8.3 g/dL (83 g/L); C-reactive protein (CRP) 2.2 mg/dL (209.53 nmol/L); and brain natriuretic peptide (BNP) 2,557 pg/mL (2,557 ng/L). Total energy intake at the time of referral was 1,450 kcal, consisting of 50 kcal from food consumed due to a severe loss of appetite, 1,200 kcal from oral nutritional supplementation, and 200 kcal from parenteral nutrition that consisted of administration of the necessary inotropes. Protein and fat intakes were 50 g and 58 g, respectively. The recommended daily allowance of micronutrients was provided by oral nutritional supplementation, with no micronutrient deficiency being noted. The patient's left ventricular ejection fraction was 23.7% and dobutamine, milrinone, and diuretics were administered.

The patient was diagnosed with malnutrition because of insufficient energy intake, weight loss, loss of muscle mass, localized or generalized fluid accumulation, and diminished functional status,[1] but he did not lose subcutaneous fat (Figure). Malnutrition occurred in the context of acute illness and/or injury due to sepsis, chronic illness due to heart failure, and social and environmental circumstances due to a severe loss of appetite.

Dual x-ray absorptiometry was used to assess the patient's appendicular skeletal muscle mass, which was 5.85 kg/m^2 and indicative of a low muscle mass when compared with the threshold value of the Asian Working Group for Sarcopenia (7.0 kg/m^2 for men).[11] The patient's Barthel Index score was 25 points, and standing training was performed during physical therapy for 20 minutes five times a week. However, gait training was not performed because of fatigability and orthostatic hypotension. Using the Asian Working Group for Sarcopenia criteria, he was diagnosed with sarcopenia because of low muscle mass and low-level physical function (usual gait speed <0.8 m/s),[11] although he was not of advanced age. In addition, the patient was diagnosed with cachexia because he sustained at least 5% weight loss in 12 months and also had a BMI <20, fatigue, anorexia, low fat-free mass index, and abnormal biochemistries,[4] although the duration of his heart failure was <3 months.

The patient's total daily energy expenditure was 2,263 kcal, based on a basal energy expenditure of 1,715 kcal calculated using the Harris-Benedict equation,[12] a gross activity factor of 1.2, and a gross stress factor of 1.2. His energy intake was planned to increase to 2,200 kcal by providing 1,600 kcal in the form of an oral nutritional supplement (protein 60 g, fat 45 g) and a meal with a dish of fruits, which the patient preferred. Protein and fat intakes were planned to increase to 88 g and 63 g, respectively. Eicosapentaenoic acid (2 g/day) was prescribed for his anorexia and cachexia.

On day 57, the patient's CRP decreased to 1.5 mg/dL (142.86 nmol/L). His oral intake at meals increased because of an improved appetite. However, his oral intake of the nutritional supplement decreased because he tired of drinking it. Therefore, his oral nutritional supplementation was decreased to 275 kcal/day, and his meal including a dish of fruits was increased to 1,850 kcal/day (protein 75 g, fat 60 g). Resistance training and stepping exercises were included in physical therapy. To maximize physical functionality, 125 mL of an oral nutritional supplement that included 200 kcal, 10 g protein, 2.5 g branched chain amino acids, and 8 g fat was consumed immediately after physical therapy.

The patient's left ventricular ejection fraction improved to 29% on day 71. Laboratory test results were as follows: serum albumin, 3.0 g/dL (30 g/L); hemoglobin, of 8.6 g/dL (86 g/L); CRP, 0.3 mg/dL (28.57 nmol/L); and BNP, 1,031 pg/mL (1,031 ng/L). The dosages of dobutamine and milrinone were decreased, and a beta-blocker and angiotensin converting enzyme inhibitor were prescribed and titrated. The walking exercise prescribed for physical therapy increased the patient's daily energy requirement to 2,640 kcal. Meals were increased to provide 2,400 kcal, and the oral nutritional supplement that was consumed immediately after physical therapy was continued.

Diagnosis	Day 43	Day 106
Malnutrition	√	
Insufficient energy intake	√	
Weight loss	√	
Loss of subcutaneous fat		
Loss of muscle mass	√	√
Localized or generalized fluid accumulation	√	
Diminished functional status	√	
Sarcopenia	√	√
Low muscle mass	√	√
Low muscle strength	NA[a]	√
Low physical function	√	
Cachexia	√	
Weight loss of at least 5% in 12 mo	√	√
BMI[b] <20	√	
Decreased muscle strength	NA	√
Fatigue	√	
Anorexia	√	
Low fat-free mass index	√	√
Abnormal biochemistry	√	

[a]NA=not available.
[b]BMI=body mass index; calculated as kg/m^2.

Figure. Diagnosis of malnutrition, sarcopenia, and cachexia.

Dobutamine and milrinone were stopped the administration on day 86 and 99, respectively.

On day 106, his body weight was 66.5 kg, and his BMI was 20.1. Laboratory tests showed: serum albumin, 4.2 g/dL (42 g/L); hemoglobin, 12.2 g/dL (122 g/L); CRP, 0.2 mg/dL (19.05 nmol/L); and BNP, 484 pg/mL (484 ng/L). Other measurements revealed the patient's daily energy intake was 2,340 kcal, his appendicular skeletal muscle mass was 6.29 kg/m^2, and his grip strength was 28 kg (right hand) and 21 kg (left