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Proses Asuhan Gizi Terstandar pada Pasien Obesitas Dengan Dekompensasi Gagal Jantung Akut Basah dan Hangat, Atrial Fibrilasi Respon Ventrikel Sedang, dan Gangguan Ginjal Akut: Sebuah Laporan Kasus

Nutrition Care Process on Obesity Patient with Acute Decompensated Heart Failure (ADHF) Wet and Warm, Atrial Fibrillation Moderate Ventricular Response, and Acute Kidney Injury: A Case Report

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ABSTRAK

Latar Belakang: *Obesitas disebabkan oleh ketidakseimbangan antara asupan energi berlebihan dan kurangnya aktivitas fisik dalam jangka waktu lama. Obesitas dapat meningkatkan risiko sindrom metabolik, termasuk risiko gagal jantung. Gagal jantung yang parah dapat meningkatkan risiko komplikasi seperti atrial fibrilasi dan gangguan ginjal akut. Sebagai langkah untuk mencegah meningkatnya tingkat keparahan gagal jantung pada pasien obesitas, diperlukan terapi diet kardiovaskular dengan prinsip cukup energi, rendah lemak, dan rendah protein yang diselenggarakan dalam rangkaian Proses Asuhan Gizi Terstandar (PAGT).*
Tujuan: *Mengetahui tatalaksana asuhan gizi terstandar pada pasien dengan pemberian diet kardiovaskuler pada pasien obesitas dengan gagal jantung, atrial fibrilasi, dan gangguan ginjal akut.*
Metode: *Studi kasus dilakukan pada bulan Februari 2023 pada pasien Rumah Sakit Khusus Infeksi (RSKI) Universitas Airlangga Surabaya*
Hasil: *Hasil evaluasi klinis menunjukkan adanya peningkatan kondisi fisik pada pasien dengan tidak adanya sesak napas dan tanda vital stabil di rentang normal pada akhir intervensi. Asupan pasien meningkat dari hari ke hari, tetapi terdapat beberapa pemenuhan zat gizi makro yang belum memenuhi target (<75%).*
Kesimpulan: *Pemulihan kondisi pasien terbilang cukup baik dengan hasil pemantauan fisik/klinis telah menunjukkan perubahan yang positif. Selain itu, pada asupan makanan terjadi peningkatan meskipun belum mencapai target asupan zat gizi makro.*

Kata kunci: *Atrial Fibrilasi, Dekompensasi Gagal Jantung Akut, Gangguan Ginjal Akut, Obesitas, Penyakit Tidak Menular*

ABSTRACT

Background: Obesity is caused by an imbalance between excessive energy intake and insufficient physical activity over a prolonged period. Obesity can increase the risk of metabolic syndrome, including heart failure. Severe heart failure can further elevate the risk of complications such as atrial fibrillation and acute kidney injury. In order to mitigate the exacerbation of heart failure severity in obese patients, it is imperative to institute a cardiovascular diet therapy employing the principles of adequate energy provision, low-fat content, and restricted protein intake within the framework of a Nutrition Care Process (NCP).
Objective: To investigate the management of standard nutritional care in obese patients with acute decompensated heart failure, atrial fibrillation, and acute kidney injury through the administration of a cardiovascular diet.

Methods: A case study was conducted in February 2023 on a patient at the University of Airlangga Infectious Disease Hospital, Surabaya.
Results: The results of the clinical evaluation demonstrated an improvement in the patient's physical condition, as evidenced by the absence of dyspnea and stable vital signs within the normal range at the end of the intervention. The patient's food intake increased progressively, although some macronutrient targets were not fully met (<75%).
Conclusion: The patient's condition showed significant improvement, as indicated by positive changes in physical/clinical monitoring. Additionally, there was an increase in food intake, although the target macronutrient intake was not fully achieved.

Keywords: Acute Decompensated Heart Failure, Acute Kidney Injury, Atrial Fibrillation, Non-Communicable Disease, Obesity

INTRODUCTION

Obesity originates from the Latin language, wherein "ob" signifies 'result of' and "esum" means 'eating'. Hence, obesity can be defined as the consequence of excessive eating patterns (Kurniagustina, 2018). According to the World Health Organization (WHO), obesity is a condition characterized by the accumulation of excessive body fat tissue (WHO, 2021). From the aforementioned definitions, it can be inferred that obesity is a medical state marked by the excessive accumulation of body fat resulting from overeating. The diagnosis of obesity can be established through the direct assessment of nutritional status using anthropometric methods such as Body Mass Index (BMI), skinfold thickness (SKF), waist-to-hip ratio (WHR), and bioelectrical impedance analysis (BIA). According to the standards set by WHO Asia Pacific, adults are categorized as obese if their BMI value exceeds 25 kg/m². For the WHR indicator, the International Diabetes Federation (IDF) designates obesity standards for adult males as >90 cm and for adult females as >80 cm (Kemenkes, 2015).

At specific waist circumference thresholds (males > 90 cm and females > 80 cm), there is a risk of increasing pro-inflammatory cytokines, leading to elevated triglycerides and decreased High-Density Lipoprotein (HDL) cholesterol levels, elevated blood pressure (hypertension), as well as diastolic dysfunction and reduced systolic function. This state is known as metabolic syndrome. Metabolic syndrome encompasses a cluster of metabolic disorders involving several conditions, including obesity, insulin resistance, impaired glucose tolerance, abnormal triglyceride levels, endothelial dysfunction, and hypertension. Each of these conditions, both individually and collectively, represents significant risk factors in the development of atherosclerosis, which can result in coronary heart disease and/or stroke (Kemenkes, 2015; Sutianingsih, 2015).

Acute Decompensated Heart Failure (ADHF) is a condition characterized by the sudden rapid worsening of heart failure (Alpert, 2015; Njoroge, 2021). Typically, this condition leads to swelling (edema) around the heart and results in breathing difficulties (dyspnea) due to the accumulation of fluid in the lungs (Alpert, 2015; Farmakis, et al., 2018). ADHF can occur in individuals who previously had chronic heart failure without symptoms (asymptomatic), wherein it leads to a sudden failure of cardiac function and often occurs in those who have not experienced heart failure before (Daryani, 2014). Obesity induces various alterations in blood flow throughout the body that affect cardiac morphology and disturb the ventricle's ability to function properly, thus increasing the risk of heart failure (Alpert, 2015). ADHF can be categorized into four hemodynamic stages based on the conditions of blood flow within organs and tissues. This classification relies on blood volume (euvolemic or "dry," and volume excess or "wet") as well as cardiac output (adequate cardiac output or "warm" and hypoperfusion or "cold") (Chaudhry, 2018; Daryani, 2021). Cardiovascular comorbidities resulting from ADHF events include hypertension, coronary artery disease, and atrial fibrillation. meanwhile, non-cardiovascular comorbidities include diabetes, renal dysfunction, chronic obstructive pulmonary disease, and anemia (Njoroge, et al., 2021).

Atrial Fibrillation is one of the most commonly encountered cardiovascular comorbidities in ADHF patients, with a prevalence of 30-40% of the total patient population (data from the European Society of Cardiology) (Mullens, 2020; Njoroge, 2021). Atrial fibrillation arises due to increased speed and irregularity of electrical signals within the atrium, resulting in rapid and irregular contractions (fibrillation). Consequently, blood accumulates in the atrium and is not adequately pumped to the ventricle. This is characterized by a very rapid heartbeat, causing the P-wave on the ECG to be absent. In this state, the atrium and ventricle do not work together as they should (Bimandoko, 2016; Damayanti, 2014). Based on ventricular response rate, Atrial Fibrillation (AF) can be categorized into

three: Fast AF response (ventricular rate = >100 beats per minute), Moderate/Normal AF response (ventricular rate = 60-100 beats per minute), and Slow AF response (ventricular rate = <60 beats per minute) (Damayanti, 2014).

Worsening renal function is a commonly encountered complication in ADHF patients undergoing treatment, even known as 'cardiorenal syndrome type I.' Approximately 20% to 40% of hospitalized ADHF patients experience concurrent kidney dysfunction (Bottiroli, 2023; Breidhardt, 2011; Mullens, 2020; Njoroge, 2021). Acute Kidney Injury (AKI) is a term synonymous with acute renal failure and is defined as a sudden decline in kidney function (glomerular filtration rate/GFR) that is transient, characterized by increased serum creatinine levels and other serum nitrogen metabolism outcomes, along with the kidney's inability to regulate fluid and electrolyte homeostasis (Rachmadi, 2011). The cardiovascular diet is a specialized dietary intervention intended for patients suffering from heart and cardiovascular diseases. This dietary approach is based on the principle of reducing saturated and trans fats intake. The primary objective of the cardiovascular diet is to meet nutritional requirements while considering the heart's capacity. In cases of Acute Decompensated Heart Failure (ADHF), particular attention is required regarding carbohydrate administration to prevent exacerbation of respiratory distress. Moreover, for patients with complicating renal dysfunction, modification of protein intake to a lower level (0.8 – 1 g/kg body weight/day) is necessary to mitigate potential renal stress (Suharyati, et al., 2019). The focus of this research encompasses the case involving an obese patient diagnosed with acute decompensated heart failure wet and warm, accompanied by complications including atrial fibrillation and acute kidney injury. The primary aim of this study is to elucidate the impact of implementing the nutrition care process therapy employing a cardiovascular diet with the principles of adequate energy provision, low-fat content, and restricted protein intake, on the progression of the patient's physiological/clinical and biochemical conditions.

METHODS

This case study was conducted in February 2023 within the Intensive Care Unit (ICU) of Airlangga University Hospital and the inpatient ward of Airlangga University Infectious disease Hospital (RSKI). Sample selection was conducted employing the purposive sampling method, with inclusion criteria consisting of inpatients with a hospitalization duration exceeding 3 days, possessing consciousness and cooperativeness, and being amenable to oral dietary intake. Meanwhile, exclusion criteria encompassed patients in critical and unconscious states, as well as those afflicted with dysphagia or limited to enteral and parenteral dietary regimens. The employed methodology involved interviews and the observation of secondary data through medical records during the assessment phase, coupled with a three-day observation period covering intake, physical/clinical aspects, and biochemical parameters. Additionally, the researcher conducted a comprehensive literature review to complement the requisite data. During the assessment session, an in-depth interview was conducted with the patient, encompassing pre-hospitalization dietary patterns utilizing the SQ-FFQ (Semi-Quantitative Food Frequency Questionnaire), the last meal consumed before hospitalization via a 24-hour food recall, daily occupational and physical activities, medical history, family history, and the patient's most recent appetite. Patient intake was monitored through the visual Comstock method, observed over a span of three days and nine meal instances. Concurrently, monitoring of the physical/clinical and biochemical domains was executed through the observation of medical records.

CASE AND DISCUSSION

The cases to be discussed in this research encompass the case of Mr. H, a 35-year-old male truck driver, who was admitted to the hospital with complaints of worsening shortness of breath since the previous day. He experiences pronounced dyspnea during light activities, necessitates sleeping with three pillows, and reports epigastric pain (+). The provisional diagnosis entails Acute Decompensated Heart Failure (ADHF) characterized as Wet And Warm, coexisting with Atrial Fibrillation (AF) Moderate Ventricular Response, alongside Acute Kidney Injury (AKI) with a differential diagnosis of Acute Chronic Kidney Disease (ACKD). The patient has a long-standing history of recurrent breathlessness and received a diagnosis of heart disease since November 2022.

Notable findings from Mr. H's physical/clinical examination include a respiratory rate of 22 breaths per minute, blood pressure of 98/76 mmHg, body temperature of 35.2℃, (+) dyspnea, and a heart rate of 118 beats per minute. Biochemical assessment reveals the following: Hemoglobin (Hb) = 16.4 g/dL, Leukocytes = 10.04 x 10^3 µL, Erythrocytes = 6.16 x 10^6 µL, Hematocrit (HCT) = 50.2%, Platelets = 309 x 10^3 µL, Mean Corpuscular Volume (MCV) = 81.5 fL, Mean Corpuscular Hemoglobin (MCH) = 26.6 pg, Mean Corpuscular Hemoglobin Concentration (MCHC) = 32.7 g/dL, Red Cell Distribution Width (RDW) = 13.4%, Mean Platelet Volume (MPV) = 9.7 fL, Lymphocytes = 31.4%, Monocytes = 8.4%, Eosinophils = 4.8%, Basophils = 0.6%, Neutrophils

= 54.8%, Plateletcrit (PCT) = 0.3%, Blood Urea Nitrogen (BUN) = 13.8 mg/dL, Creatinine (Cr) = 1.4 mg/dL, Estimated Glomerular Filtration Rate (eGFR) = 62 mL/min/1.73 m², Fasting Blood Glucose Level = 124 mg/dL, High-Sensitivity Troponin I (HS Troponin I) = 21.6 ng/L, Albumin = 3.89 g/dL, Sodium (Na) = 136 mmol/L, Potassium (K) = 4.3 mmol/L, Chloride (Cl) = 105 mmol/L. Mr. H experienced a decline in appetite upon admission to the hospital. Mr. H's measured body weight was 90 kg, and his height was 165 cm. The prescribed maximum daily fluid intake for Mr. H was set at 1500 mL based on the Doctor's recommendation.

As a truck driver, Mr. H has a habit of eating outside of his home. His dietary history reveals frequent consumption of high-saturated fat and coconut milk-laden dishes such as rendang, kikil (tripe), offal, and fried foods. Additionally, he has a preference for high-sugar beverages like soda, es teler (mix fruit and jelly with syrup and sweet condensed milk), and es campur (mix fruit with syrup and sweet condensed milk). Mr. H is allergic to chicken, dislikes meat after experiencing his medical condition, and prefers fish as his animal protein source. He had previously attempted dieting after his heart disease diagnosis but found the restrictions too discomforting due to gastric distress. He neither engages in exercise, nor smokes, nor consumes alcohol.

The patient's prolonged consumption of high-fat saturated coconut milk-based foods without regular exercise has resulted in the accumulation of adipose tissue, thereby increasing the risk of obesity. Furthermore, the patient's penchant for packaged and processed beverages high in sugar and fat exacerbates the excess deposition of adipose tissue (Kemenkes, 2015; Dinicolantonio, et al., 2016). This surplus accumulation of adipose tissue leads to an increase in lean body mass and central and overall blood volume production (Alpert, 2015). The elevated blood volume production is indicated by Mr. H's higher erythrocyte count than the normal level. These mechanisms collectively stimulate an increase in cardiac output, facilitated by a reduction in systemic blood vessel resistance. While the heart rate remains relatively stable, the amplified cardiac output is predominantly attributed to an augmented volume of blood flow in the left ventricle. Given the relatively consistent heart rate, the increased cardiac output is primarily attributed to an escalated volume of blood ejected from the left ventricle. In uncomplicated obesity, the heightened cardiac output seemingly leads to left ventricular dilation and eccentric left ventricular hypertrophy (LVH). LVH in obese individuals can induce diastolic dysfunction, and in severe cases, contribute to left ventricular heart failure, alongside venous and pulmonary artery hypertension (Alpert, 2015).

Left ventricular weakness results in the regurgitation of blood back into the atrium, and subsequently into the pulmonary circulation, right ventricle, and right atrium. This condition, along with hypoxemia due to sleep apnea and hypoventilation often seen in severe obesity, contributes to the development of right ventricular heart failure (Alpert, 2015). As blood is not optimally pumped out of the right side of the heart, it begins to accumulate in the peripheral venous system. Consequently, the outcome is a diminishing blood volume within the circulation, a decline in cardiac output and blood pressure, and an exacerbation of the heart failure cycle (Daryani, 2014). This condition is characterized by elevated levels of High Sensitivity Troponin I (HS-TnI), a biomarker indicating myocardial necrosis, exceeding the normal range. Furthermore, conditions of both left and right ventricular heart failure can arise, partly due to the involvement of proinflammatory cytokines, such as tumor necrosis factor (TNF), transforming growth factor-β (TGF-β), interleukins (IL)-6, and IL-1(Njoroge, et al., 2021)[28-29]. The activity of cytokines in the pathophysiology of Acute Decompensated Heart Failure (ADHF) is indicated by elevated monocyte and eosinophil levels in the patient, surpassing the normal range. Additionally, acute disturbances in ADHF hinder compensatory mechanisms, leading to an increase in left or right (or both) ventricular filling pressures, culminating in symptoms experienced by Mr. H, including dyspnea, palpitations (high heart rate), and other signs of decompensation, such as elevated respiratory rate and low body temperature (Mullens, et al., 2020).

In patients with Acute Decompensated Heart Failure (ADHF), atrial fibrillation ensues due to heightened pressure and stress exerted on the ventricular walls, leading to disruptions in atrial electrical conduction. Consequently, irregular and rapid contractions (fibrillation) occur (Damayanti, 2014). Moreover, the acute reduction in cardiac output precipitates compromised organ perfusion, consequently promoting target organ damage, notably the kidneys (Njoroge, et al., 2021). Kidneys are notably sensitive to abrupt declines in cardiac output (CO), and acute renal hypoperfusion leads to an immediate decrease in glomerular filtration rate, urine production, and parenchymal oxygenation. These mechanisms contribute to the occurrence of Acute Kidney Injury (AKI), and if chronic, elevate the risk of Chronic Kidney Disease (CKD) (Bottiroli, 2023; Breidhardt, 2011).

In this particular case, the patient was in a cooperative and bed rest condition, necessitating the use of the most recent data available in the medical records for weight and height measurements, which yielded a recorded body weight of 90 kg and a height of 165 cm. Nutritional status was determined through the Body Mass Index (BMI), resulting in a value of 33 kg/m². Based on these findings, the patient was categorized as having Obese II

according to the classification provided by the WHO Asia-Pacific guidelines. Furthermore, insights derived from an in-depth interview revealed that the patient had experienced a diminished appetite prior to hospitalization, attributed to breathing difficulties. The patient also exhibited a historical dietary pattern characterized by the consumption of high-sugar and saturated-fat foods and beverages, coupled with a deficient dietary intake prior to hospital admission.

Table 1. Results of In-Depth Dietary Pattern Interview Prior to Hospitalization Using SQ-FFQ Method

Food Types	Frequency		Food Types
	≥ 1x/day	1-3x/weeks	
Carbs	White rice (2x)		2 full rice ladles (150 g)
Animal Proteins		Beef soto, beef tendon, curry, offal (alternately)	1 cup (150 g)
Plant Proteins	Tofu, tempeh (2x)		2 medium pieces (40 g)
Vegetables	Vegetable stew (2x)		1 cup (100 g)
Fruits		Apple, pear, papaya, (alternately 3x)	1 fruit (100-120 g)
Beverages	Mineral water, Pocari Sweat (1x)	Ice teler, soda, Mixed fruit ice (3x alternately)	1 small bottle 330 mL (Soda & Pocari), 1 cup of ice
Snacks		Sari roti sandwich (3x)	1 piece (50 g)

Based on the results of the dietary pattern interview using the SQ-FFQ method (Table 1), it is evident that Mr. H has a habit of consuming foods and beverages high in saturated fats, characterized by a propensity for indulging in dishes and drinks rich in coconut milk, organ meats, deep-fried items, and excessive sugar content. Mr. H's occupation as a truck driver fosters the practice of acquiring food and beverages from outside sources, thus enhancing his motivation to consume such items. Excessive consumption of saturated fats can elevate total cholesterol levels, thereby increasing an individual's susceptibility to coronary heart disease. Furthermore, a prolonged high-sugar diet may lead to glucose intolerance and insulin resistance, promoting the onset of hyperglycemia. Hyperglycemia resulting from sugar consumption can induce proinflammatory and prothrombotic effects, glycation of LDL, lipogenesis, dyslipidemia, and oxidative stress (Dinicolantonio, et al., 2016).

The interview findings also reveal that the patient experienced his first heart attack after consuming two servings of tripe stew in November 2022. This inappropriate dietary habit was attributable to a lack of prior nutritional education for the patient.

Tabel 2. Results of Patient Assessment Evaluation

Examination	Results	Normal Value	Interpretation
Prior Hospitalizations Intake (Food recall)			
Energy	1571,7 kcal	2458,9 kcal	Severe Deficit (63,9%)
Protein	50,6 g	92,2 g	Severe Deficit (54,8%)
Fat	21,7 g	68,3 g	Severe Deficit (31,8%)
Carbohydrate	285,3 g	368,8 g	Moderate Deficit (77,4%)
Anthropometry			
Height	165 cm		
Body Weight	90 kg		
BMI	33 kg/m^2	18,5-22,9 kg/m^2	Obese II
Biochemistry Blood Test			
Hb	16,4 g/dL	13,2 - 17,3 g/dL	Normal
Leukocytes	10,04 x 10^3 µL	6-12 x 10^3 µL	Normal
Erythrocytes	6,16 x 10^6 µL	4,4-5,9 x 10^6 µL	Higher
HCT	50,2%	40 - 52%	Normal
Platelets	309 x 10^3 µL	150-440 x 10^3 µL	Normal
MCV	81,5 fL	80 - 100 fL	Normal
MCH	26,6 pg	26 - 34 pg	Normal
MCHC	32,7 g/dL	32 - 36 g/dL	Normal
RDW	13,4%	11,5 - 14,5%	Normal
MPV	9,7 fL	6,8 - 10 g/dL	Normal
Lymphocytes	31,4%	25 - 40%	Normal
Monocytes	8,4%	2 - 8%	Higher

Examination	Results	Normal Value	Interpretation
Eosinophils	4,8%	2 - 4%	Higher
Basophils	0,6%	0 - 1%	Normal
Neutrophils	54,8%	50 - 70%	Normal
PCT	0,3%	0,2-0,36%	Normal
BUN	13,8 mg/dL	8 - 18 mg/dL	Normal
Cr	1,4 mg/dL	0,6 - 1,1 mg/dL	Higher
eGFR	62 mL/min/1,73 m^2	≥90 mL/min/1,73 m^2	Lower
FBS	124 mg/dL	70 - 99 mg/dL	Higher
HS Troponin I	21,6 ng/L	<2 ng/L	Higher
Albumin	3,89 g/dL	3,4 - 4,8 g/dL	Normal
Na	136 mmol/L	135 - 147 mmol/L	Normal
K	4,3 mmol/L	3,5 - 5 mmol/L	Normal
Cl	105 mmol/L	98 - 108 mmol/L	Normal
Physical/Clinical			
Cardio-Pulmonary System	Shortness of Breath		Experiencing shortness of breath
Heart Rate	118 bpm	80-100 bpm	Higher
Respiratory rate	22 bpm	12-20 bpm	Higher
Temperature	35,2 ℃	36-37,5℃	Lower
Blood Pressure	98/76 mmHg	90-120/60-80 mmHg	Normal

Based on the 24-hour dietary recall interview utilizing the food recall method (Table 2), it is discerned that the patient exhibits deficiencies in energy, protein, fat, and carbohydrate intake. The adequacy level of energy intake is classified based on the percentage of energy fulfillment concerning individual daily requirements, as stipulated by the Indonesian Ministry of Health (2003), encompassing four levels: severe deficit (<70% fulfillment), moderate deficit (70-79% fulfillment), mild deficit (80-89% fulfillment), normal (90-110% fulfillment), and excess (>110% fulfillment). The patient has experienced a decreased appetite since before hospitalization due to shortness of breath, yet there is no history of weight loss. There is no prior medical history for the patient, and no family history of illnesses is evident.

Based on the laboratory findings provided by the hospital, it is evident that the patient has elevated levels of erythrocytes, monocytes, eosinophils, serum creatinine, HS Troponin I, and fasting blood glucose. Conversely, it is also observed that the patient's eGFR is lower than normal, indicating a reduction in kidney function (Bottiroli, 2023; Breidhardt, 2011). In individuals with ADHF, the increase in erythrocyte count occurs as a compensatory mechanism due to chronic hypoxia or when cells and body tissues experience oxygen deficiency, thereby stimulating heightened erythropoietin production and inducing the bone marrow to generate more erythrocytes (Gangat, 2021; Gherasim, 2019; Kodliwadmath, et al., 2020). This is manifested by the patient's reported shortness of breath and increased respiratory rate, which contributes to the occurrence of hypoxia.

The increase in monocytes and eosinophils in ADHF patients occurs due to myocardial fibrosis. Myocardial fibrosis reduces left ventricular (LV) elasticity and increases filling pressure and atrial load. This leads to fibrosis in the LV and atrium, resulting in the development of Atrial Fibrillation, thus perpetuating a vicious cycle (Seguela, et al., 2015; Shahid, 2018). The elevation in serum creatinine levels and decrease in eGFR occur due to reduced kidney function resulting from acute hypoperfusion due to decreased cardiac output, leading to diminished renal blood flow (Bottiroli, 2023; Breidhardt, 2011). The elevation of HS Troponin I indicates heart damage. Prolonged ischemia induces myocardial necrosis and membrane disruption, leading to the release of structural and cytosolic troponins into the bloodstream at higher-than-normal levels. Myocardial ischemia arises from increased oxygen demand or decreased oxygen supply within the body. Reduced myocardial oxygen supply can result from arrhythmia and arterial hypotension (Gherasim, 2019). This is marked by an elevated heart rate and reduced body temperature in the patient.

The objective of providing dietary intervention to the patient is to maintain the body weight, prevent weight loss, reduce excessive fat intake to alleviate cardiac strain, ensure adequate nutritional intake without burdening cardiac and renal functions, as well as avert fluid retention and maintain normal electrolyte levels (Suharyati, et al., 2019). The dietary principles provided to the patient involve adequate energy and low-fat consumption. Furthermore, the nutritional diagnosis encompasses four distinct diagnoses spanning three domains: intake, clinical, and behavior. Within the intake domain, the diagnoses are as follows: 1. (NI-2.1) Inadequate oral intake (P) related to the nutrition requirements knowledge deficit (E), as evidenced by severe deficits in energy intake (63.9%), protein

(54.8%), and fat (31.8%), as well as moderate deficiency carbohydrate intake (77.4%) (S); 2.) Decreased fat, fluid, and sodium needs (P) related to Heart Failure (E), as evidenced by an elevated HS Troponin I level (21.6 ng/L) exceeding normal levels (S). In the clinical domain, the diagnosis is Obesity type II (P) related to a nutrition knowledge deficit (E), as evidenced by a BMI of 33 kg/m^2 and a history of high-fat and sugar consumption (S). Finally, in the attitude domain, the diagnosis is (NB-1.1) Food/nutrition-related knowledge deficit (P) related to no prior cardiac nutrition-related education (E) as evidenced by prior unsuccessful diet attempts causing discomfort and gastric distress (S).

According to recommendations from The Academy's Evidence Analysis Library (EAL), the calculation of the patient's energy requirement can employ the Mifflin-St Jeor formula, utilizing the actual body weight if direct calorimetry-based basal metabolic rate calculation is not feasible. Additionally, in overweight and obese patients, estimating energy needs using adjusted body weight or ideal body weight via the Harris-Benedict formula carries a high risk of underestimation, up to 42% (Kohn, 2015). The patient's nutritional needs were calculated using the Mifflin-St Jeor formula, utilizing the actual body weight of 90 kg, with physical activity factor for bedrest at 1.2 and moderate stress factor at 1.4, with a 500 kcal reduction in total energy expenditure to prevent overestimation risk (Mifflin, 1990). The total energy requirement for Mr. H was determined as 2,458.9 kcal per day. Based on the food recall results, the patient's intake in the last 24 hours before hospitalization was 1,571.7 kcal or 63.9% of the daily requirement, indicating a severe energy deficit.

The calculation for protein requirement was set at 15% of total energy, equivalent to 92.2 g with 50% being high biological value protein sources, such as low-fat animal protein (Mafrici, et al., 2021; Suharyati, et al., 2019). In patients with low albumin levels, a high-protein diet may be necessary. However, in cases of heart failure with concomitant renal complications, protein intake should be adjusted based on the severity of the complications. In Mr. H's case, given the acute kidney injury, a sufficient protein diet is recommended, with a protein intake of 0.8 - 1.0 g/kgBW/day or 10-15% of the total energy requirement (Suharyati, et al., 2019). For heart failure patients, saturated fat, carbohydrate-derived triglycerides, and cholesterol intake need to be restricted. Greater emphasis is placed on the consumption of unsaturated fats to mitigate the risk of exacerbating the heart condition. The patient's fat requirement was set at 25% of total energy, with monounsaturated fat at 10% and polyunsaturated fat at 5% of total energy. Additionally, a limit on saturated fat intake was imposed, not exceeding 10% of total energy (Suharyati, et al., 2019). The total daily fat requirement provided was 68.3 g.

Carbohydrate provision requires careful consideration in heart failure patients, as excessive intake can worsen respiratory distress (Suharyati, et al., 2019). Recommended carbohydrate intake ranges from 50-60% of total energy, sourced from complex carbohydrates (such as rice, grains, corn, sweet potatoes, etc.) while limiting simple carbohydrates (such as sugar, syrups, honey, and related products). For Mr. H, carbohydrate intake was set at 60% of total energy, amounting to 368.8 g[30]. Fluid intake was restricted to 1500 mL/day as per doctor's recommendations. In heart failure patients with acute kidney injury complications, fluid administration must be tailored to fluid balance. Dietary intervention was gradually introduced to the patient each day, aimed at helping the patient adapt to the prescribed diet portions. The percentage of dietary intervention on day 1 was set at 80% of the plan, day 2 at 90%, and day 3 at 100%, in accordance with the patient's calculated needs.

Table 3. Three Days Dietary Intake Monitoring Results

Date	Observation	Energy (kcal)	Protein (gram)	Fat (gram)	Carbs (gram)
Day-1 4/2/23	Planning	1967.1	73.8	54.6	295.0
	Intake	**1416.3**	**50.2**	**42.3**	**203.0**
	%Intake Fulfillment	70.2%	68.0%	77.4%	68.8%
Day-2 5/2/23	Planning	2213	83	61.5	331.9
	Intake	**1845.7**	**71.8**	**54.7**	**267.2**
	%Intake Fulfillment	83.4%	86.5%	89.0%	80.5%
Day-2 5/2/23	Planning	2458.9	92.2	68.3	368.8
	Intake	**2323.7**	**85.1**	**67.5**	**349.3**
	%Intake Fulfillment	94.5%	92.3%	98.8%	94.7%

Based on Table 3, it can be observed that the patient's intake during the three-day intervention exhibited an increase each day. On the first day, the patient's protein and carbohydrate intake were still categorized as severely deficient, while energy and fat intake were moderately deficient. On the second day, there was an improvement in the patient's intake, moving to a mild deficiency in energy and all macronutrients. By the third day, the patient's intake had reached a normal range for energy and all macronutrients (Depkes RI, 1996). Thus, it can be concluded that the patient's dietary adjustment and adaptation during the three-day intervention were successful, with the patient meeting the planned nutritional requirements on the final day of the intervention.

Table 4. Blood Test Biochemistry Monitoring Results

Indicators	Assessment	Results		
		Day-1 Intervention	Day-2 Intervention	Day-3 Intervention
Erythrocytes	6,16 x 10^6 µL	No further measurements were conducted	-	No further measurements were conducted.
BUN	13,8 mg/dL		-	
Cr	1,4 mg/dL		-	
eGFR	62 mL/mm		-	
Albumin	3,89 g/dL		-	
Na	136 mmol/L		136 mmol/L	
K	4,3 mmol/L		4 mmol/L	
Cl	105 mmol/L		98 mmol/L	

Table 5. Physical/Clinical Monitoring Results

Indicators	Assessment	Results		
		Day-1 Intervention	Day-2 Intervention	Day-3 Intervention
Cardio-Pulmonary System	Shortness of Breath	No more shortness of breath	No more shortness of breath	No more shortness of breath
Heart Rate	118 bpm	102 bpm	126 bpm	78 bpm
Respiratory rate	22 bpm	20 bpm	20 bpm	18 bpm
Temperature	35,2℃	36℃	36℃	36,1℃
Blood Pressure	98/76 mmHg	105/75℃	116/87℃	106/74℃

Based on Table 4, further biochemical measurements were conducted on the second day of the intervention, but not all indicators were examined. Some measurable indicators include Na, K, and Cl, which remained stable within the normal range from the first day of assessment to the second day of intervention. Monitoring the levels of Na, K, and Cl in the patient aims to ensure that electrolyte levels remain stable throughout the intervention period.

Based on Table 5, it is evident that all measurable indicators remained within the normal range during the course of the intervention. In terms of the "shortness of breath" indicator, Mr. H did not experience shortness of breath again after the initial assessment until the last day of the intervention. Fluctuations were observed in the "heart rate" indicator, with an increase on the second day and a subsequent decrease below the normal threshold on the final day. Gradual progress toward normal levels was observed in the "respiratory rate" and "temperature" indicators from the first to the last day of the intervention, while the "blood pressure" indicator remained consistently within the normal range from the initial assessment to the last day of the intervention.

CONCLUSION

Based on the results of the three-days monitoring and evaluation of nutritional intervention, it was observed that there was an increase in intake from the first to the second day, which was still categorized as deficient, and by the third day, the planned nutritional requirements were successfully met. In the biochemical domain, the examination results on the last day of the intervention successfully achieved the goal, with stable levels of Na, K, and Cl within the normal range. In the physical/clinical domain, there was an improvement in the patient's physical condition from the beginning to the end of the intervention, although the patient's heart rate indicator remained below the normal range.

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Author Contributions

EMO*:* writing-original draft, methodology, investigation, formal analysis; NYR: supervision, writing-review & editing; F: supervision; TM: supervision.

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