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CASE REPORT

NUTRITIONAL THERAPY IN A GERIATRIC PATIENT WITH ISCHEMIC STROKE, CHRONIC KIDNEY DISEASE, PNEUMONIA, AND DIABETES MELLITUS

(TERAPI GIZI PADA PASIEN GERIATRI DENGAN STROKE ISKEMIK, PENYAKIT GINJAL KRONIS, PNEUMONIA, DAN DIABETES MELITUS)

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ABSTRACT

Stroke patients are often at risk of malnutrition and dehydration, conditions that can be exacerbated by comorbidities such as chronic kidney disease (CKD), pneumonia, and type II diabetes mellitus (DM II). Nutritional therapy is essential to improve nutritional status and support clinical and metabolic recovery in these patients. The aim of this article is to report the application and outcomes of nutritional therapy in a 62-year-old female patient with ischemic stroke, CKD, pneumonia, and type II diabetes mellitus. This is a case report of a 62-year-old woman diagnosed with ischemic stroke, pneumonia, CKD, and DM II. The patient presented with loss of consciousness (GCS score E2M2V2), left hemiparesis, and edema in the arms, sacral area, and pretibial region. Nutritional therapy was initiated with an initial caloric intake of 800 kcal, gradually increased to a target of 1800 kcal/day, tailored to the patient's clinical condition and laboratory findings. The nutritional therapy began with 800 kcal/day and was gradually increased. Although the patient's serum albumin levels improved from 2.49 g/dL to 2.85 g/dL, her kidney function continued to decline, with increasing levels of blood urea nitrogen, creatinine, C-reactive protein (CRP), and procalcitonin (PCT). The patient ultimately experienced cardiac arrest on the 29th day of treatment, and the family declined resuscitation efforts. Despite the nutritional therapy, the patient's metabolic abnormalities could not be fully corrected without hemodialysis, which was declined by the family. The therapy provided some support but was insufficient to prevent the progression of renal failure and the patient's subsequent death.

Keywords: chronic kidney disease, nutritional therapy, pneumonia, stroke, type II diabetes mellitus

ABSTRAK

Pasien stroke sering kali berisiko mengalami malnutrisi yang diperburuk dengan penyakit penyerta seperti penyakit ginjal kronis (PGK), pneumonia, dan diabetes melitus (DM). Terapi nutrisi sangat penting untuk meningkatkan status gizi dan mendukung pemulihan klinis dan metabolik pada pasien. Laporan kasus ini mengenai seorang wanita berusia 62 tahun yang didiagnosis dengan stroke iskemik, pneumonia, CKD, dan DM tipe II. Pasien datang dengan penurunan kesadaran (skor GCS E2M2V2), hemiparesis kiri, dan edema di lengan dan pretibial. Terapi nutrisi dimulai dengan asupan kalori awal 800 kkal yang secara bertahap ditingkatkan hingga target 1800 kkal/hari, target protein 36-40 gram, dan suplementasi cholecalciferol 1x/hari. Meskipun kesadaran, kadar serum albumin, dan gula darah pasien sempat ada perbaikan, fungsi ginjalnya terus menurun, dengan peningkatan kadar ureum, kreatinin, c reactive protein (CRP), dan procalcitonin (PCT). Keluarga tidak setuju untuk dilakukan hemodialisa. Pasien akhirnya meninggal dunia akibat komplikasi penyakit yang dialaminya. Terapi nutrisi memberikan perbaikan sementara tetapi tidak cukup untuk mencegah progresivitas gagal ginjal dan gangguan metabolik pada pasien.

Kata kunci: chronic kidney disease, diabetes type II, pneumoni, strok, terapi gizi

INTRODUCTION

Stroke is the second leading cause of death worldwide, with a global incidence of 12.2 million cases and a prevalence of 101 million in 2019.[1] In Indonesia, the prevalence of stroke increased significantly from 7 per 1,000 population in 2013 to 10.9 per 1,000 population in 2018.[2] Beyond neurological damage, stroke often leads to comorbidities such as chronic kidney disease (CKD) and pneumonia.[3] A Polish study found 66.4% of ischemic stroke patients developed renal function decline within four months.[3] The area of the brain that received stroke is a relevant factor related to the use of mechanical ventilators, especially when it affects the brain areas that regulate consciousness (thalami, limbic system, and formation reticularis in the brainstem), breathing (respiration centers in the cortex, pons, and medulla) and swallowing (medulla and brainstem) which may increase the risk of respiratory failure.[4] Stroke associated pneumonia (SAP) is also common, worsening outcomes in patients with dysphagia or requiring ventilation.[5] Although endotracheal tubes reduce large volume aspiration, they cannot fully prevent micro aspiration of pharyngeal or gastric contents, which may contribute to complications, especially among malnourished patients.[6,7]

Malnutrition is a major problem that often occurs in stroke patients, with a prevalence ranging from 19-72%.[8] Studies conducted in South Korea and Bandung reported malnutrition in 58.9–78.9% of patients with acute or subacute stroke, and 51.9% of patients overall, particularly those aged ≥ 65 years.[9] Metabolic disorders such as diabetes mellitus (DM) can further worsen

nutritional status in stroke Diabetes Mellitus (DM).[10]

Malnutrition is closely associated with an increased risk of complications, including hypoproteinemia (serum albumin level < 35 g/dL), which increases susceptibility to infection and worsens clinical outcomes, ultimately contributing to a higher risk of mortality.[11]

This study aims to report the application of nutritional therapy in a 62-year-old female patient with stroke and comorbidities of CKD, pneumonia and type II diabetes mellitus.

MATERIALS AND METHODS

1. Research Design and Location

This case report employed a descriptive design to analyze nutritional therapy in a 62-year-old female with **ischemic stroke**, CKD, pneumonia, and type II diabetes mellitus. The study was conducted at Intan Husada Hospital, Garut, Indonesia, and covered the patient's care period from May 24 to June 16, 2024.

2. Population and Sample

The subject of the study was a 62-year-old female patient admitted to the intensive care unit (ICU) with a diagnosis of multiple lacunar infarcts, CKD, pneumonia, and type II diabetes mellitus. This study received ethical approval from the Ethics Committee of Intan Husada Hospital with approval number: No:012/PNT/DIR/RSIH/VIII/2024.

3. Data Collection

Variables studied: the main variables observed were nutritional status, blood glucose, electrolytes, albumin, blood urea nitrogen (BUN), creatinine, and related biomarkers in response to nutritional therapy.

Instruments and materials used: Instruments included anthropometric measurements (arm circumference, height) for nutritional status assessment and laboratory tests (haematology, albumin, electrolytes, blood glucose, estimated glomerular filtration rate (eGFR), C-reactive protein (CRP), procalcitonin (PCT), BUN, and creatinine) for biochemical assessment. BUN and creatinine levels during hospitalization are summarized in Figures 1–4.

Study procedures: The patient received parenteral and enteral nutrition using an MCT-containing, kidney-specific formula, starting at 800 kcal per day and gradually increased to 1,800 kcal/day, with added cholecalciferol. Monitoring included nutritional intake, vital signs, and changes in biomarker.

RESULTS

Clinical Course

a. Day 1

A 62-year-old woman presented with loss of consciousness following a one-week history of vomiting and fever. She had a prior stroke (one year earlier) and more than three years of hypertension and type II diabetes mellitus without regular follow-up, though she had remained functionally independent post-stroke. On examination, she was sopor (GCS E2M2V2) with bilateral rhonchi, pretibial edema, arm edema, and left hemiparesis. Anthropometric assessment showed a height of 155 cm, mid-upper arm circumference (MUAC) of 23.7 cm, estimated weight of 70 kg, and an ideal body weight (IBW) of 63 kg. Investigations revealed cardiomegaly with bronchitis on chest X-ray, multiple lacunar infarcts in the basal ganglia and periventricular white matter on CT scan of the brain, and drainage of 500 cc of black fluid following nasogastric tube insertion. Initial vital signs and laboratory findings are summarized in Table 1**.**

Table 1 Initial vitals and labs

Parameter	Result	Normal Range
Blood Pressure (BP)	139/84 mmHg	<140/90 mmHg
Heart Rate (HR)	109 bpm	60–100 bpm
Respiratory Rate (RR)	22 /min	12–20 /min
Temperature (Temp)	36.2 °C	36–37.5 °C
Oxygen Saturation (SpO_2)	88%	>94%
Hemoglobin (Hb)	10.1 g/dl	12–16 g/dl
White Blood Cells (WBC)	37,580/µl	4,000–10,000/µl
Platelets	208,000/µl	150,000–450,000/µl
Sodium (Na)	150.3 mmol/L	135–145 mmol/L
Potassium (K)	4.6 mmol/L	3.5–5.0 mmol/L
Random Blood Sugar (RBS)	150 mg/dl	70–140 mg/dl
HbA1c	5.06%	<6.5%
Blood Urea Nitrogen (BUN)	77 mg/dl	7–20 mg/dl
Creatinine	1.49 mg/dl	0.6–1.2 mg/dl
Albumin	—	3.5–5.9 g/dl

b. Day 2

Despite the administration of oxygen at 15 L/min via a non-rebreather mask, the patient's SpO_2 continued to decline, necessitating intubation and transfer to the intensive care unit (ICU).

c. Day 7

Serum albumin was measured at 2.1 g/dL, indicating severe hypoalbuminemia. The family was advised to consider hemodialysis for the

patient; however, they declined.

d. Day 8

A repeat chest X-ray revealed findings with bronchopneumonia.

e. Day 9

C-reactive protein (CRP) levels were measured on day 9 of treatment, with a value >200 **mg/L** (normal reference range <**5 mg/L**).

f. Day 11

On day 11, hemoglobin declined to 6.6 g/dL, necessitating transfusion of two units of packed red blood cells. Inflammatory markers also worsened, as evidenced by elevated CRP levels and a further increase in the white blood cell (WBC) count. Concurrently, fasting blood glucose rise to 316 mg/dL, leading to the initiation of an insulin infusion with monitoring every 4 hours. A tracheostomy was subsequently performed to replace the endotracheal tube.

g. Day 12

Laboratory tests showed a hemoglobin (Hb) level of 8.1 g/dL and a markedly elevated procalcitonin level of 96.0 ng/mL, consistent with severe infection.

h. Day 13

Albumin slightly increased to 2.9 g/dL.

i. Day 14

On day 14, the patient was referred to a clinical nutritionist, and nutritional therapy was reassessed. Caloric intake was gradually increased to 569.4 kcal/day (Figure 1). The Subjective Global Assessment (SGA) classified the patient as "C" (severe protein-energy malnutrition). Enteral nutritionconsisted of an MCT formula (3 × 125 kcal) and a kidney-specific formula (2 × 150 kcal), supplemented with a 200 ml/day parenteral solution (172 kcal, 17.9 g protein) (Figure 2). The patient's level of consciousness remained stable, though aphasia persisted.

j. Day 15

Nutritional therapy was initiated under the guidance of a clinical nutritionist, targeting 1,800 kcal/day and 36 - 40 g of protein. Actual intake reached 994.6 kcal (Figure 2). However, tolerance issues limited full delivery. Neurological status showed slight improvement, with spontaneous eye opening but persistent aphasia. Blood glucose levels stabilized below 250 mg/dL, allowing the monitoring frequency to be reduced to every 8 hours. Hemoglobin levels increased to 8.6 g/dL

k. Day 16

The enteral formula was adjusted to include a medium-chain triglyceride (MCT) formula (3 × 125 kcal) and a kidney-specific formula (2 × 150 kcal) to improve tolerance.

l. Day 18

On day 18, On day 20, a gastric residue of 300 mL led to temporary cessation of feeding, reducing caloric intake to 323 kcal/day (Figure 1). Micronutrient supplementation, including cholecalciferol 5000 IU/day, was initiated.

m. Day 19

On day 19 Procalcitonin decreased to 5.21 ng/mL, suggesting partial infection control.

n. Day 21

Despite ongoing nutritional and medical support, renal function progressively deteriorated. Blood urea nitrogen (BUN) and creatinine levels continued to rise, while the estimated glomerular filtration rate (eGFR) declined to 14 mL/min/1.73 m² (Figure 4). The family continued to refuse dialysis.

o. Day 22

The patient's neurological status deteriorated, with decreasing consciousness and increasing somnolence. Blood glucose leves increased again, ranging 274 to 362 mg/dL, requiring intensification of insulin therapy. Nutritional efforts continued, gradually increasing caloric intake, to 569.4 kcal/day (Figure 1).

p. Day 29 (Outcome)

Despite comprehensive medical and nutritional interventions, the patient ultimately succumbed to progressive renal failure and complications from infection on the 29th day of hospitalization.

Table 2 Glucose level during hospitalization

Date (2024)	Monitoring Frequency	Range (mg/dl)	Interpretation
June 4	4-hourly	316–424	Markedly elevated, >250 mg/dl
June 5	4-hourly	183–256	Improved but fluctuating, some >250
June 6	4-hourly	232–299	Mostly >250
June 7	4-hourly	144–303	Variable, occasional >250
June 8	4-hourly	139–222	Controlled (<250)
June 9	4-hourly	165–257	Borderline, one value >250
June 10	4-hourly	107–219	Within target
June 11	4-hourly	121–220	Within target
June 12	4-hourly	127–161	Well controlled
June 13	4-hourly	126–230	Borderline, one high value
June 14	8-hourly	177–182	Controlled
June 15	6-hourly	100–362	Marked fluctuations, >250 at times
June 16	6-hourly	112-176	Well controlled

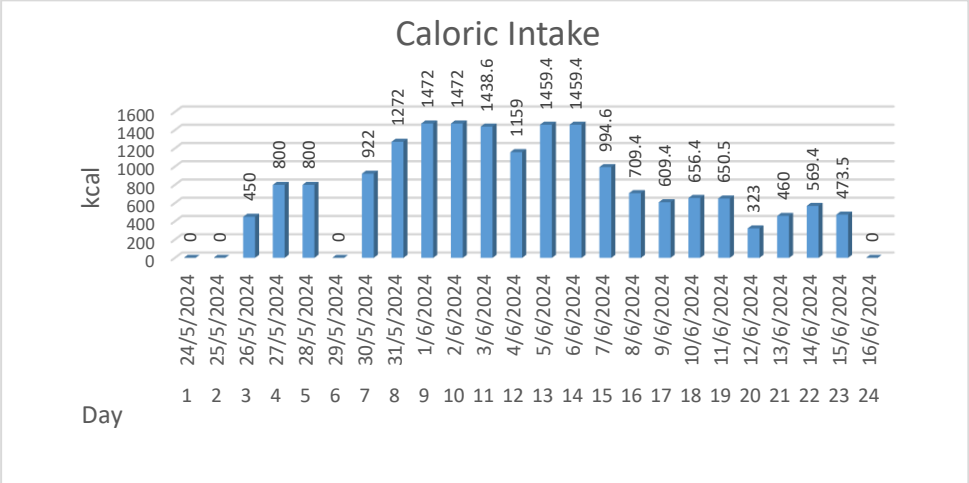

Figure 1 Caloric intake during hospitalization**.**

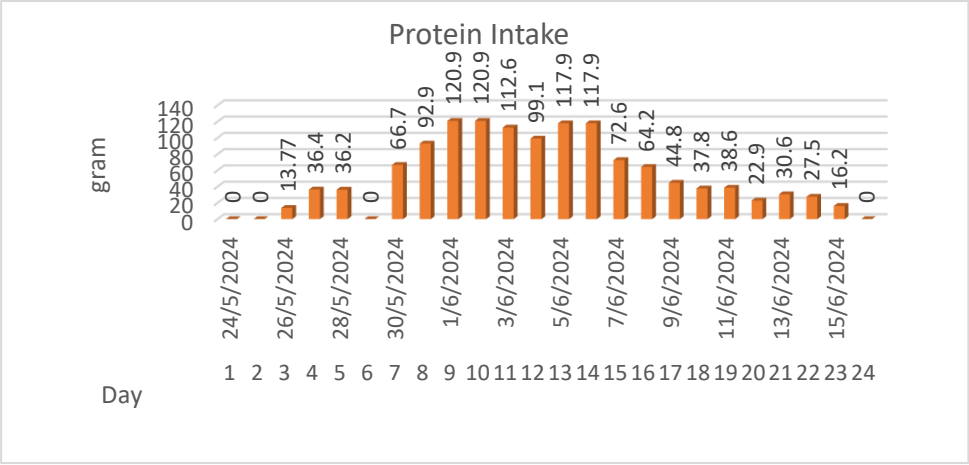

Figure 2 Protein intake during hospitalization.

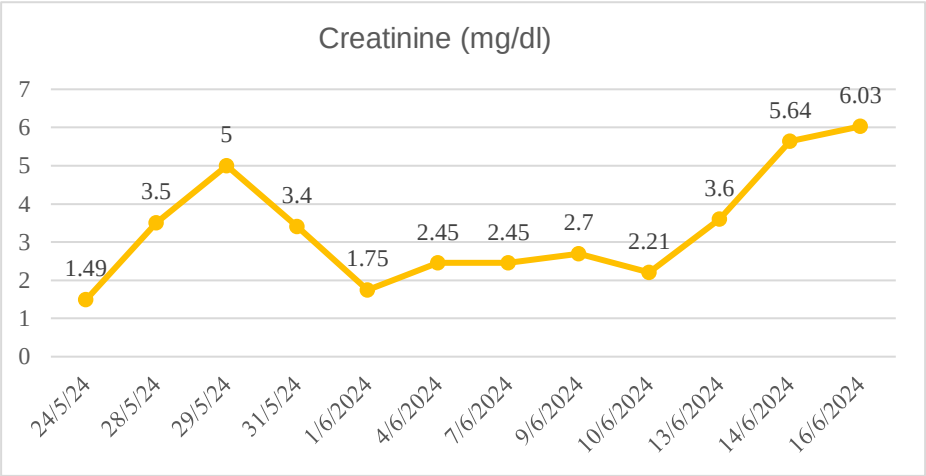

Figure 3 Creatinine level during hospitalization.

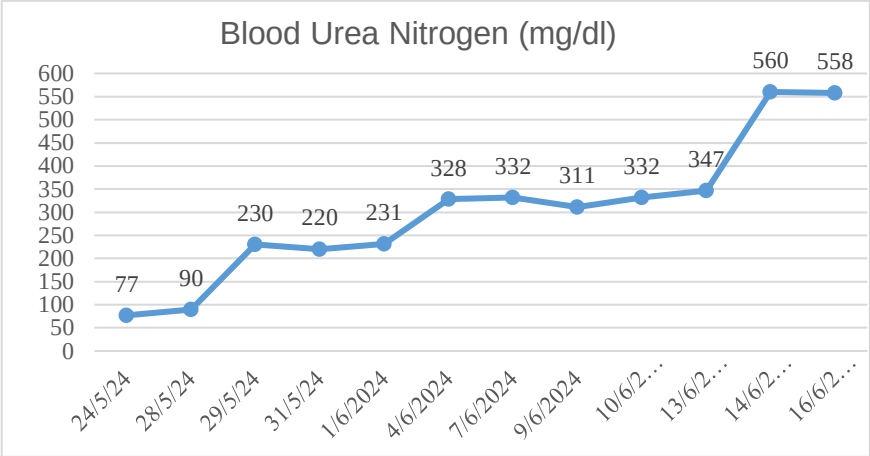

Figure 4 Blood urea nitrogen level during hospitalization.

DISCUSSION

In this patient, malnutrition developed due to reduced intake caused by impaired consciousness, recurrent vomiting, and large gastric residues, compounded by increased energy and protein demands from stroke-related inflammation, comorbidities, and invasive procedures.[9,12] Nutritional assessment using the Subjective Global Assessment (SGA) classified her as having severe protein-energy malnutrition (Score C), consistent with clinical findings of edema, vomiting, black nasogastric tube (NGT) residue, and comorbidities including diabetes, pneumonia, and CKD. Laboratory tests confirmed hypoalbuminemia, reflecting both inadequate intake and inflammation-related catabolism. [12]

Energy requirements were carefully adjusted considering kidney dysfunction and comorbidities, with a target of 1,800 kcal/day, aligning with KDOQI recommendations of 30–35 kcal/kg/day for CKD patients.[13] Energy intake must be individualized based on age, sex, activity, body composition, CKD stage, degree of inflammation, and comorbid conditions, while balancing the risk of protein–energy wasting and the need for protein restriction. In this case, approximately 50% of energy was derived from carbohydrates, with the remainder from protein and fat. [14]

The American Diabetes Association (ADA) and Kidney Disease: Improving Global Outcomes (KDIGO) recommend a protein intake of approximately 0.8 g/kg body weight per day for patients with CKD.[15] In this patient, the protein target was set at 36–40 g/day, adjusted for progressive CKD without dialysis and persistent hyperglycemia. This follows KDOQI guidelines recommending 0.6–0.8 g/kg/day for patients with stage 3–5 CKD and diabetes, aiming to support glycemic

control while minimizing additional renal burden.[15]

The administration of medium-chain triglyceride (MCT)-containing diet was selected to provide readily available energy without exacerbating the patient's metabolic condition, particularly in relation to glycemic control in type II diabetes mellitus.[16] Renal-specific formulas were also prescribed because they contain lower levels of sodium, potassium, magnesium, and phosphorus compared with standard formulas, thereby helping to electrolyte imbalance and fluid overload.[17]

In patients with stroke, malnutrition is a significant risk factor associated with poorer prognosis, especially when accompanied by comorbidities such as CKD, diabetes mellitus, and pneumonia.[6, 8, 9] Previous studies have demonstrated that CKD is common among stroke patients and may worsen clinical outcomes due to heightened inflammation and metabolic complications.[3]

Cholecalciferol supplementation was administered for its potential anti-inflammatory and antioxidant effects. The patient experienced recurrent gastric residue, necessitating temporary suspension and gradual reintroduction of enteral feeding in smaller volumes.[18] Despite incremental increases, the nutritional target could not be fully achieved due to progressive clinical deterioration. According to the European Society for Clinical Nutrition and Metabolism (ESPEN), enteral nutrition should only be resumed once bleeding has ceased and there are no signs of rebleeding.[19]

In this patient, hypoalbuminemia developed as a result of comorbid conditions that triggered systemic inflammation and increased metabolic demands, while recurrent gastric residues further restricted nutritional intake. Albumin deficiency may disrupt coagulation balance, promote thrombosis, worsen stroke outcomes, and increase the risk of complications such as pneumonia.[11]

Electrolyte imbalances were likely multifactorial, arising from inadequate intake, fluid restriction implemented for edema management, and neuroendocrine disruption associated with acute stroke. Fluctuations in sodium levels may have been influenced by medical therapies, including intravenous fluids, antibiotics, and mannitol, which can affect vasopressin secretion. Additionally, comorbid diabetes mellitus and pneumonia may have contributed indirectly to electrolyte instability. Progressive renal dysfunction further impaired potassium excretion, leading to potassium disturbances.[20, 21]

Anemia is common in patients with CKD and typically worsens as renal function declines. Chronic inflammation contributes to iron deficiency and impaired

absorption, thereby disrupting hemoglobin synthesis. Despite blood transfusions and ongoing nutritional therapy, recovery of haemoglobin levels remained limited.[22]

Elevated leukocyte counts, C-reactive protein (CRP), and procalcitonin (PCT) levels reflected systemic inflammation associated with stroke, pneumonia, and diabetes mellitus. This finding is consistent with previous studies demonstrating an association between increased CRP levels, reduced estimated glomerular filtration (eGFR), and lower serum albumin concentrations.[23] Although PCT levels decreased, renal deterioration continued, as evidenced by increased blood urea nitrogen and creatinine levels.

The primary goal of nutritional therapy was to minimize nitrogen waste accumulation in CKD, which can promote muscle protein catabolism and perpetuate chronic inflammation. Progressive renal dysfunction further restricted protein intake, complicating achievement of protein targets.[24, 25] Although, nutritional therapy supported improvement in nutritional status,it was insufficient without definitive treatments. This case underscores the importance of multidisciplinary management and more aggressive therapeutic interventions in patients withcomplex conditions.

Further research is required to establish effective nutritional protocols for patients with CKD who are not receiving renal replacement therapy, including more intensive or combined strategies aimed at preserving renal function and reducing mortality.

CONCLUSION

Nutritional therapy may contribute to stabilization of clinical status, improved glycemic control, increased serum albumin levels, and partial improvement in selected biomarker parameters. However, in patients with advanced chronic kidney disease, nutritional intervention alone is insufficient to halt progressive renal deterioration. Definitive management, including hemodialysis when indicated, remains essential to prevent further kidney damage.

CONFLICT OF INTEREST

The authors declare that no conflict interests related to this study.

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