Medical nutrition therapy in hemodynamically unstable patients due to cardiogenic shock with infected bronchiectasis and severe protein-energy malnutrition[☆]

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ABSTRACT

Objective: Cardiogenic shock is defined as tissue hypoperfusion due to cardiac dysfunction. It is associated with hemodynamic unstability and elevated arterial lactate as one indicator for anaerobic metabolism. Hypercatabolic state in this condition leads to increasing nutritional requirement and negative nitrogen balance. Therefore, medical nutrition therapy by considering metabolic tolerance can prevent further metabolic deterioration and loss of lean mass and improve the patient's clinical outcome.

Methods: A 44-years-old female patient with severe protein-energy malnutrition (*Subjective Global Assessment* Score C; MUAC 15 cm) suffered from hemodynamic unstability due to cardiogenic shock and infected bronchiectasis at the infection center of Wahidin Sudirohusodo Hospital. Intake was postponed due to mean arterial pressure 56 mmHg on vasopressor support and oxygen saturation below 93%. Physical examinations showed loss of subcutaneous fat, lung crackles and wheezing, muscle wasting, and pretibial edema. Laboratory assessments showed elevated arterial lactate (3.2 mmol/L), hypoalbuminemia (2.4 g/dL), lymphocytopenia (650/μL), elevated liver enzymes (SGOT 780 U/L; SGPT 868 U/L), and urine urea nitrogen (5 g/24 h). Nutritional therapy was started after mean arterial pressure ≥65 mmHg with a stable dosage of the vasopressor drug and decreased arterial lactate level to 2.2 mmol/L then given gradually with a target calorie of 1500 kcal and protein 1.5–1.8 g/kg ideal body weight/day using high protein diet. Arterial lactate and blood gas analyses were controlled every day to determine the target of nutritional therapy day by the day. Snakehead fish extract, zinc, vitamin B complex, Thiamine, vitamin C, vitamin A, vitamin D3, and Curcumin were supplied.

Result: After 15 days of nutritional therapy, the patient was discharged from the hospital with stable hemodynamic without vasopressor support, adequate nutritional intake, improvement of anthropometric parameters, and laboratory test results (arterial lactate 1.6 mmol/L, albumin 3.1 g/dL, lymphocyte 1.871/μL, SGOT 34 U/L, SGPT 41 U/L, urine urea nitrogen 0.72 g/24 h).

Conclusion: Adequate nutritional therapy, which is planned by evaluating hemodynamic tolerance, can improve patient clinical outcomes and positive nitrogen balance in the hemodynamically unstable patient.

Introduction

Cardiogenic shock is associated with hemodynamic unstable and elevated arterial lactate as one indicator for anaerobic metabolism due to low perfusion in tissue.[1,2] Evaluation of hemodynamic condition can be used to determine a plan of nutritional therapy.[3] Nutritional therapy consists of macronutrients and micronutrients requirements. In the critically ill patient with hypercatabolic state, nitrogen losses can reach 16–20 g/day and, in some cases, up to 24 g/day compared to those without catabolic stress which generally loses only 10–12 g of nitrogen/day.[4] Loss of body mass around 30–40% will increase the mortality rate due to starvation or protein malnutrition.[5] High protein diet and adequate micronutrients intervention can improve the outcome of the patient. Delayed nutritional therapy will worsen negative nitrogen balance, nutritional status, and hypercatabolic state. On the other hand, too early nutritional intervention can worsen anaerobic metabolism in a hemodynamically unstable patient. Therefore, the nutritional goal can be planned by evaluating hemodynamic changes to determine the time for postponing nutritional therapy or increasing the nutritional target.

Methods

This is a case report of a patient who was treated in September 2018 at the Wahidin Sudirohusodo Hospital. This case report has fulfilled the requirement of the Ethics Committee of Hasanuddin University School of Medicine. The patient and the family were given informed consent and agreed to be reported as a case report without mentioning the name and private information.

Calorie Intake

Day	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15
Calorie Intake (kcal)	0	300	490	490	720	1012	1158	1408	1557	1557	1650	1600	1708	1775	1725
Calorie Target (kcal)	0	450	450	750	750	1125	1125	1500	1500	1500	1900	1900	1900	1900	1900

Fig. 1. Calorie intake of the patient.

Protein Intake

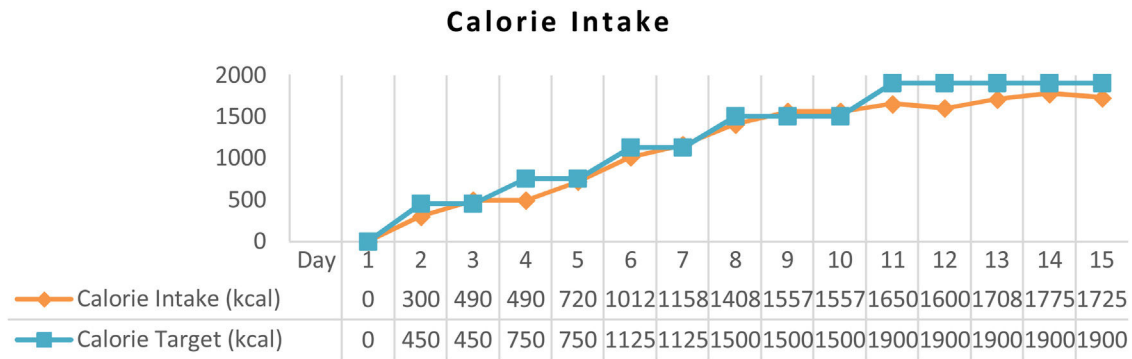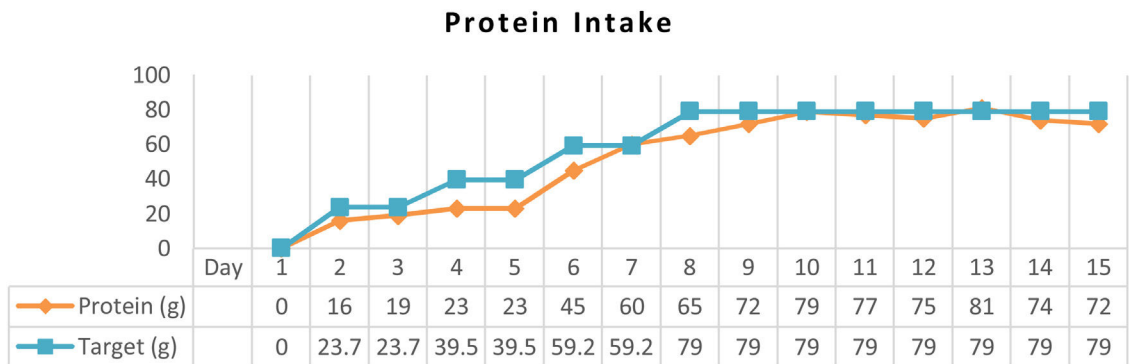| Day	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15
Protein (g)	0	16	19	23	23	45	60	65	72	79	77	75	81	74	72
Target (g)	0	23.7	23.7	39.5	39.5	59.2	59.2	79	79	79	79	79	79	79	79

Fig. 2. Protein intake of patient.

A 44-years-old female patient with severe protein-energy malnutrition with the marasmic type (*Subjective Global Assessment* Score-C; MUAC 15 cm) was consulted from Pulmonology Departement with hemodynamic unstability due to cardiogenic shock and infected bronchiectasis since one day ago at the infection center of Wahidin Sudirohusodo Hospital, Makassar. She had a history of low dietary intake since 3 months ago due to decreased appetite and shortness of breath. Unintentional weight loss of about 4 kg was present 3 months ago. Intake was postponed due to mean arterial pressure 56 mmHg on vasopressor support and oxygen saturation below 93%. Physical examinations showed loss of subcutaneous fat, lung crackles and wheezing, muscle wasting, and pretibial edema. Laboratory assessments showed elevated arterial lactate (3.2 mmol/L), hypoalbuminemia (2.4 g/dL), lymphocytopenia (650/μL), elevated liver enzymes (SGOT 780 U/L; SGPT 868 U/L), and urine urea nitrogen (UUN 5 g/24 h). Echocardiography showed the left and right ventricle (ejection fraction 64%) with mild diastolic dysfunction of the left ventricle. Abdominal ultrasonography showed congestive liver signs. Thoracal MSCT showed dilated bronchiectasis.

Basal metabolic rate (BMR) by Harris-Benedict Formula was 983 kcal, and total energy expenditure (TEE) was 1500 kcal. Nutritional therapy was started after mean arterial pressure ≥ 65 mmHg with a stable dosage of the vasopressor drug for 6 h and arterial lactate 2.2 mmol/L with 30–40% of TEE on 2–3 days after stable hemodynamic. Macronutrients were given for patients with protein 0.8–1.3 g/ideal body weight (IBW)/day, carbohydrates 45–50%, and lipid 30% for seven days after hemodynamic unstability onset. Protein was gradually increased to 1.5–1.8 g/IBW/day using a high protein diet and high branched-chain amino acid formula to create a positive nitrogen balance based on the UUN result. The positive nitrogen balance target was +4. In addition, routine arterial lactate and blood gas analyses were assessed to control hemodynamic tolerance.
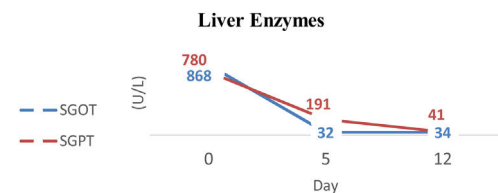
Liver Enzymes

Fig. 3. Improvements of liver enzymes.
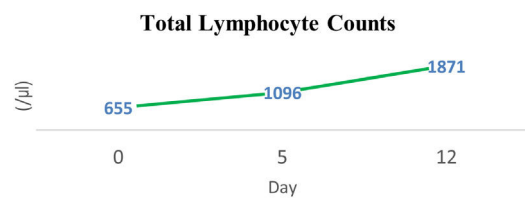
Total Lymphocyte Counts

Fig. 4. Improvements of total lymphocyte counts.

Result

After arterial lactate was below 2.0 mmol/L with tappering-off vasopressor dosage, nutritional therapy was planned for 100% TEE on day 8 (Figs. 1 and 2). In addition, supplementations of Snakehead fish extract, zinc 20 mg, vitamin B complex, thiamine 300 mg, vitamin A 6000 IU, vitamin C 500 mg, and vitamin D3 400 IU were given to improve immune and metabolic function. After 15 days of nutritional therapy, the patient was discharged from the hospital with stable hemodynamic without vasopressor support, adequate oral intake (90–95% of TEE), improvement of anthropometric parameters, and laboratory test results (arterial lactate 1.6 mmol/L, albumin 3.1 g/dl, lymphocyte 1.871/μL, SGOT 34 U/L, SGPT 41 U/L, UUN 0.72 g/24 h) (Figs. 3 and 4).

Metabolic Changes After Onset Of Cardiogenic Shock

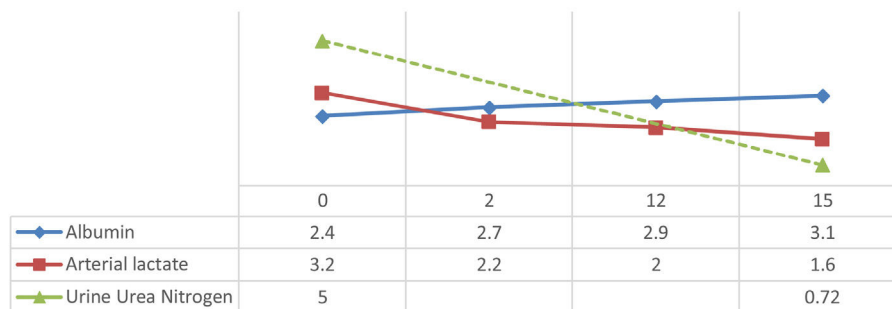|	0	2	12	15
Albumin	2.4	2.7	2.9	3.1
Arterial lactate	3.2	2.2	2	1.6
Urine Urea Nitrogen	5			0.72

Fig. 5. Metabolic changes after nutritional intervention.

Discussion

The patient was admitted to the emergency department with diagnosis of cardiogenic shock (Fig. 5). She had mean arterial pressure of 56 mmHg on vasopressor support, oxygen saturation below 93%, and elevated arterial lactate level to 3.2 mmol/L. Hyperlactatemia in this patient was hyperlactatemia type A that occurred due to impaired tissue perfusion.[6,7] Furthermore, periodic control of arterial lactate level was carried out to assess the hemodynamic status to determine nutritional therapy target.[8] Cuthberson, the first person who described the metabolic changes that occurred after major trauma in 1942, divided metabolic response to trauma into Ebb and Flow Phases.[9] The Ebb phase begins within the first hour after trauma and can last up to 24–48 h, characterized by a decrease in total energy requirements and a decrease in urinary nitrogen excretion. Targets in this phase are hemodynamic adaptation and stable tissue perfusion. The flow phase is also known as the catabolic phase. The metabolic response in this phase is mediated by catabolic hormones (such as glucagon, catecholamines, and corticosteroids) and accompanied by insulin resistance. Acute phase protein synthesis in liver, pro-inflammatory cytokines and free radicals are also increase in this phase.[10] Hyperlactatemia in critically ill patients and particularly those in shock is generally interpreted as a marker of secondary anaerobic metabolism due to inadequate oxygen supply inducing cellular distress.[2] In this case, arterial lactate test was performed 18 h (3.2 mmol/L) after the onset of shock, then re-assessed 24 h later (2.2 mmol/L), and controlled every day till lactate level 1.6 mmol/L to plan 100% nutritional target. Mizuki et al. reported lactate clearance as one of the most important predictors of in-hospital mortality in patient treated with extracorporeal cardiopulmonary resuscitation after cardiac arrest.[11]

In the critical phase, the priority of protein synthesis in liver is to form acute-phase proteins, such as C-reactive protein, α1-acid glycoprotein, α1-protease inhibitor, fibrinogen, and haptoglobin, than constitutive proteins, such as albumin, prealbumin, and retinol-binding protein.[4] Albumin level in this patient was 2.4 g/dL at admission and gradually increased up to 3.1 g/dL, which was correlated with the decrement of UUN from 5 g/24 h to 0.72 g/24 h (Fig. 5). Loss of lean mass about 30–40% due to negative nitrogen balance and undernourishment are correlated with high mortality rates.[5,12] Data from an observational study showed that protein intake of 1.2–1.5 g/kg IBW/day was associated with a better outcome. Ishibasi et al. showed that protein intake of 1.5 g/kg BW/day was associated with a minimal negative nitrogen balance.[13] Negative nitrogen balance (−2.89) in this patient was calculated from UUN (5 g/24 h) by using a nitrogen balance correction formula in critical patients (nitrogen balance = (protein intake/6.25) − (UUN/0.85) − 4).[5] Therefore, the protein target was increased to 1.8 g/kg IBW/day to achieve positive nitrogen balance

(+0.5) using high protein formula.[14] After 15 days of nutritional therapy, UUN was 0.72 g/24 h with a higher estimated positive nitrogen balance (+4.72). Positive nitrogen balance (around +4 g/day) can support protein synthesis by 25 g/day and increase body mass up to 100 g/day.[10]

Elevated liver enzymes can be caused by acute liver injury due to hepatic hypoperfusion or due to acute cardiogenic liver injury. This condition is commonly seen in a patient with decreased cardiac output, cor pulmonale disease, and acute decompensated heart failure. In acute circulation disruption, blood flow to the liver decreases about 10% for every 10 mmHg decrease in arterial pressure.[3] The role of Branched Chain Amino Acids (BCAAs) in acute liver injury is still controversial. There is no recommendation about it. However, several animal studies have shown the role of BCAAs in animals experiencing an acute liver injury. Kitagawa et al. reported that BCAAs play a role in improving the liver condition in mice that experience acute liver injury due to the ischemic–reperfusion phase through improved microcirculation, inflammatory response, and leukocyte adhesion.[15] Daily Snakeheadfish extract, zinc 20 mg, vitamin B complex, Thiamine 300 mg, vitamin C 500 mg, vitamin A 6000 IU, vitamin D3 600 IU, and Curcumin 1.200 mg were given. Vitamin D has a role in modulating innate and adaptive immune responses.[16] Snakehead fish extract contains high protein, albumin, and several micronutrients.[17,18] This extract increases albumin levels in hypoalbuminemia patients and modulates the immune response by molecular and immunological mechanisms.[19] Zinc plays a role in boosting the immune system by optimizing A20mRNA upregulation and decreasing NF-κB activation, suppressing activation of TNF-α, IL-1β, and IL-8. Zinc supplementation can reduce oxidative stress.[20] Parikh et al. reported that vitamin C 5 mg/kg/day in children or 200 mg/day in adults could optimize mitochondrial function and reduce arterial lactate level.[21] *In vitro* studies demonstrate that T cell development requires vitamin C, while vitamin C also enhances T cell proliferation and may influence T cell function.[22]

Conclusion

Adequate medical nutrition therapy, which is planned by evaluating hemodynamic tolerance, can improve clinical outcomes and positive nitrogen balance in hemodynamically unstable patients.

Conflicts of interest

The authors declare no conflict of interest.

References

1. Frost P, Wise MP. Recognition and early management of the critically ill ward patient. Br J Hosp Med. 2007;68 Suppl. 10:M180–3.

2. Levy B. Lactate and shock state: the metabolic view. Curr Opin Crit Care. 2006;12:315–21.

3. Preiser J, Zanten A, Berger M, et al. Metabolic and nutritional support of critically ill patients: consensus and controversies. Crit Care. 2015;19:1–11.

4. Golts E, Choi J, Wilson W, et al. Nutritional and metabolic evaluation and monitoring. In: Wilson WC, Grande CM, Hoyt DB, editors. Critical care. Boca Raton, FL: CRC Press; 2007. p. 569–82.

5. Dickerson RN. Using nitrogen balance in clinical practice. Hosp Pharm. 2005;40:1081–7.

6. Reddy AJ, Lam S, Bauer S, et al. Lactic acidosis: clinical implications and management strategies. Cleve Clin J Med. 2015;82:615–24.

7. Singer P, Blaser A, Berger M, et al. ESPEN guideline on clinical nutrition in the intensive care unit. Clin Nutr. 2019;38:48–79.

8. Flordelís Lasierra JL, Pérez-Vela JL, Montejo González JC. Nutrición enteral en el paciente crítico con inestabilidad hemodinámica. Med Intensiva. 2015;39:40–8.

9. Stahel PF, Flierl MA, Moore EE. Metabolic staging after major trauma – a guide for clinical decision making? Scand J Trauma Resusc Emerg Med. 2010;18:18–20.

10. Şimşek T, Şimşek HU, Cantürk NZ, et al. Response to trauma and metabolic changes: posttraumatic metabolism. Ulus Cer Derg. 2014;30:153–9.

11. Mizutani T, Umemoto N, Taniguchi T, et al. The lactate clearance calculated using serum lactate level 6 h after is an important prognostic predictor after extracorporeal cardiopulmonary resuscitation: a single-center retrospective observational study. J Intensive Care. 2018;6:1–7.

12. Taslim NA, Virani D, Sumartini NK, et al. Energy regulation in newly diagnosed TB with chronic energy deficiency: free fatty acids and RBP4. Asia Pac J Clin Nutr. 2017;26:S73–8.

13. Ishibashi N, Plank L, Sando K, et al. Optimal protein requirements during the first 2 weeks after the onset of critical illness. Crit Care Med. 1998;26:1529–35.

14. Bukhari A, Taslim NA, As'ad S, et al. Comparison of different early enteral feeding formulas on critically ill patients. J Nutr Sci Vitaminol (Tokyo). 2020;66:S2–10.

15. Kitagawa T, Yokoyama Y, Kokuryo T, et al. Protective effects of branched-chain amino acids on hepatic ischemia–reperfusion-induced liver injury in rats: a direct attenuation of Kupffer cell activation. Am J Physiol Liver Physiol. 2013;304:G346–55.

16. Aranow C. Vitamin D and the immune system. J Rheumatol. 2010;37:491–5.

17. Fauzan MR, Dahlan CK, Taslim NA, et al. The effect of giving fish extract (Pujimin Plus) on intake of protein and hemoglobin hypoalbuminemic patients. Enfermería Clínica. 2020;30:452–5.

18. Fatma N, Metusalach, Taslim NA, et al. The protein and albumin contents in some species of marine and brackishwater fish of South Sulawesi, Indonesia. AACL Bioflux. 2020;13:1976–85.

19. Wijaya I, Taslim NA, Natzir R, et al. Molecular and immunological mechanisms of channa striata in diabetic wound healing. Int J Pharm Res. 2020;12(sp2).

20. Prasad AS. Zinc in human health: effect of zinc on immune cells. Mol Med. 2008;14(5–6):353–7.

21. Parikh S, Saneto R, Falk MJ, et al. A modern approach to the treatment of mitochondrial disease. Curr Treat Options Neurol. 2009;11:414–30.

22. van Gorkom G, Wolterink RK, van Elssen C, et al. Influence of vitamin C on lymphocytes: an overview. Antioxidants. 2018;7:41.