CASE REPORT

Nutrition Therapy in Post-Hartmann's Procedure Patients in The Intensive Care Unit

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ABSTRACT

Background: The Hartmann procedure is a surgical procedure for treating colorectal cancer that is widely used in emergencies because it is fast and has a minimal risk of anastomotic leakage. This procedure is usually performed on rectosigmoid cancer. Colorectal cancer patients who undergo gastrointestinal surgery are considered at risk of malnutrition, so adequate nutritional therapy is needed.

Case: We report a 51-year-old male patient who came to the intensive care unit (ICU) with an unconscious condition, was intubated, received Norepinephrine support, and had a reddish black product in the nasogastric tube (NGT). The patient was referred with a diagnosis of post-operative Hartmann's procedure day-3 (D-3) for indications of high obstructive ileus due to rectosigmoid tumor, septic shock, acute kidney injury (AKI), and peptic ulcer. During treatment in the ICU, the patient received antibiotic therapy (Meropenem and Metronidazole), proton pump inhibitors (PPIs), and parenteral nutrition via a central venous catheter (CVC).

Discussion: Parenteral nutrition was given from the beginning of admission to the ICU because oral and enteral nutrition could not be provided due to gastrointestinal bleeding (peptic ulcer). Moreover, the patient was considered at risk of malnutrition with evidence of critical illness more than 48 hours post-gastrointestinal surgery due to cancer. Also, there was a post-operative fasting period, usually for several hours to 1-2 days, depending on the patient's condition.

Conclusion: Nutritional therapy, as part of the management of critically ill patients, should be given at the right time, in the most effective way, and in appropriate doses for each individual to avoid malnutrition during treatment.

Keywords: acute kidney injury; Hartmann's procedure; parenteral nutrition; peptic ulcer; septic shock

INTRODUCTION

Cumulative genetic changes in the colon and rectum's epithelial cells cause excessive cell division, migration, and differentiation, as well as cell proliferation, invasion, and metastasis, which leads to colorectal cancer.[1] As the second most lethal and third most often diagnosed cancer worldwide, colorectal cancer, which includes cancer of the colon and/or rectum, is a serious health issue.[2]

In 2020, colorectal cancer accounted for 10% of the global cancer incidence and 9.4% of cancer deaths, second only to lung cancer at 18%.[3] Globally, the estimated incidence rate of colorectal cancer was 19.5%. 34,189 cases (8.6%) of colorectal cancer have been reported in Indonesia.[1] Globally, there are 50,000 new instances of anal cancer, 0.7 million new cases of rectal cancer, and 1.15 million new cases of colon cancer, according to GLOBOCAN figures. The increase in the incidence of colorectal cancer is due mainly to increased exposure to environmental risk factors due to shifts in lifestyle and diet toward Westernization.[3]

The most common therapy for colorectal cancer, particularly in stages 0 to II, is surgery.[2] Hartmann's procedure is still widely used in emergencies because it is a quick operation, and the risk of anastomotic leakage can be avoided. This procedure is usually performed in patients with rectosigmoid cancer.[4,5]

Early nutrition provision in postlaparotomy patients provides the advantage of reducing the risk of sepsis up to threefold compared with delaying nutrition provision. Nutrition can be provided immediately after hemodynamics are stable. Nutrition provision in the first 48 hours post-laparotomy is associated with decreased infection rates, mortality, and length of hospitalization.[6,7,8]

Oral nutrition, if possible, is the next choice, and early enteral nutrition, which gradually increases in the first 48 hours. If neither is possible, parenteral nutrition can be given. The recommended time for providing parenteral nutrition is if, in the first 7 days, the patient cannot tolerate enteral nutrition, even though actions have been taken to optimize the provision of enteral nutrition.[9–12]

Studies have shown that oral nutritional intake is often insufficient after gastrointestinal surgery, especially in patients with cancer. The causes of insufficient post-operative oral nutritional intake are a lack of knowledge about nutritional management and a post-operative fasting period. The causes of the patients' symptoms include abdominal discomfort due to nausea, bloating, pain, and other psychological factors.[13] The purpose of this case report is to determine the benefits and procedures for providing nutritional therapy to post-Hartmann's procedure patients in the intensive care unit (ICU).

CASE

We report a 51-year-old man referred from another hospital with complaints of decreased consciousness and post-Hartmann's procedure day-3 (D-3), septic shock (SOFA score 9), acute kidney injury (AKI), peptic ulcer, and hypokalemia. Three days before entering the hospital, the patient was initially diagnosed with a high obstructive ileus due to a rectosigmoid tumor suspected to be malignant. Initially, he complained of difficult defecation accompanied by difficulty passing gas, abdominal pain, and distension. After surgery, the

patient's condition declined, and he was then treated in the ICU. During treatment in the ICU, the patient received therapy in the form of intravenous fluid drug (IVFD) NaCl 0.9% 10 ml/hour, Meropenem 1 gr/8 hours iv, Metronidazole 500 mg/8 hours iv, Fluconazole 200 mg/24 hours iv, Esomeprazole titration 8 mg/hour, Furosemide titration 5 mg/hour, Fentanyl titration 30 mcg/hour, Norepinephrine titration 0.3 mcg/kg/minute, Paracetamol 1 gr/8 hours iv and Vitamin K 10 mg/8 hours iv. Patients also receive hemodialysis with Baxter Prismaflex (Baxter International, Inc., USA), which was performed for 3 hours with bicarbonate-free heparin dialysate with indications of AKI. The patient had no prior medical history of conditions such as asthma, diabetes mellitus, hypertension, cardiovascular disease, or chronic kidney disease (CKD).

A primary survey in the emergency unit was conducted, and the general condition of the patient was found to be unconscious. The airway was clear, with an endotracheal tube (ETT) size 7.5mm installed at a depth of 20cm with ventilator mode pressure support inspiratory mandatory (PSIMV) with respiratory rate 12 times/minute, PEEP 5, FiO_2 80%. The respiratory rate was 20x/minute, SpO_2 was 98% in ventilator mode setting, and basic vesicular breath sounds were heard in both lung fields, with no additional breath sounds. During circulation, the blood pressure was 108/70 mmHg on 0.05 mcg/kg/minute of Norepinephrine, the heart rate was 90 times/minute, the pulse was strong enough, the extremities were warm, and the capillary refill time (CRT) was less than 2 seconds. On examination of disability and exposure, a Glasgow coma scale (GCS) E2M4Vet was obtained; the isochoric pupil was 3 mm/3 mm, the light reflex was normal, and the temperature (T) was 36.7 °C.

On physical examination of the abdomen, there were positive bowel sounds, tenderness on surgical wounds, a stoma on the left abdomen filled with brownish product, a drain on the right abdomen containing serous hemorrhagic product, and the nasogastric tube (NGT) product was reddish black. Other physical examinations were within normal limits. The patient's anthropometric status was 64 kg in body weight, 167 cm in body height, and 22.9 kg/m^2 in body mass index (BMI) (good nutritional status).

Based on the results of laboratory tests on the first day of treatment, azotemia and hypokalemia were detected.

The patient underwent blood gas analysis (BGA), and partially compensated metabolic acidosis with increased lactate levels was found.

Table 1. Results of laboratory examination and blood gas analysis (BGA)

Parameter	Value (normal value)	Parameter	Value (normal value)
Complete Blood Count (CBC)	Electrolyte		
Eritrosit	3.84x10^6/uL (3.65-5.05x10^6/uL)	Na	136 mmol/L (136-145 mmol/L)
Hemoglobin	11.0 g/dL (10.4-16.0 g/dL)	K	2.9 mmol/L (3.5-5.1 mmol/L)
Hematokrit	34.0% (35.0-51.0%)	Cl	104 mmol/L ((98-107 mmol/L)
Leukosit	9.500x10^3/uL (6.0-18.0x10^3/uL)	BGA	
Trombosit	147.000x10^3/uL (150-450 x10^3/uL)	pH	7.001 (7.35-7.45)
PCT	1.35 ng/mL (<0.55 ng/mL)	pCO_2	30.5 mmHg (35-45 mmHg)
BUN	100 mg/dL (4-19 mg/dL)	pO_2	136.7 mmHg (75-100 mmHg)
Creatinin	9.09 mg/dL (0.16-0.39 mg/dL)	BE	-23.04 mmol/L (-2-2 mmol/L)
Random Blood Sugar (RBG)	230 mg/dL (<200 mg/dL)	HCO^3	7.5 mEq/L (22-26 mEq/L)
PT	14.7/11.0 s (9.4-12.5 s)	Lactate	6.0 mmol/L (<2 mmol/L)
APTT	33.5/31.2 s (25.1-36.5 s)		
INR	1.34 (0.90-1.10)		
Albumin	1.99 g/dL (3.80-5.40 g/dL)		
SGOT	1926 u/L (7-50 u/L)		
SGPT	657 u/L (5-56 u/L)		

The patient underwent a chest X-ray examination. The resultsrevealed that the lungs and heart were within normal limits, an ETT was installed on the tracheal projection with the distal end facing caudally at a height of approximately 3.62 cm above the carina, a CVC was installed through the right subclavian vein and the distal end facing caudally at the cavo-atrial junction projection at the level of intercostal space (ICS) 5-6, and a hemodialysis cath was installed with insertion through the right jugular vein and the distal end facing caudally at the right atrium projection.

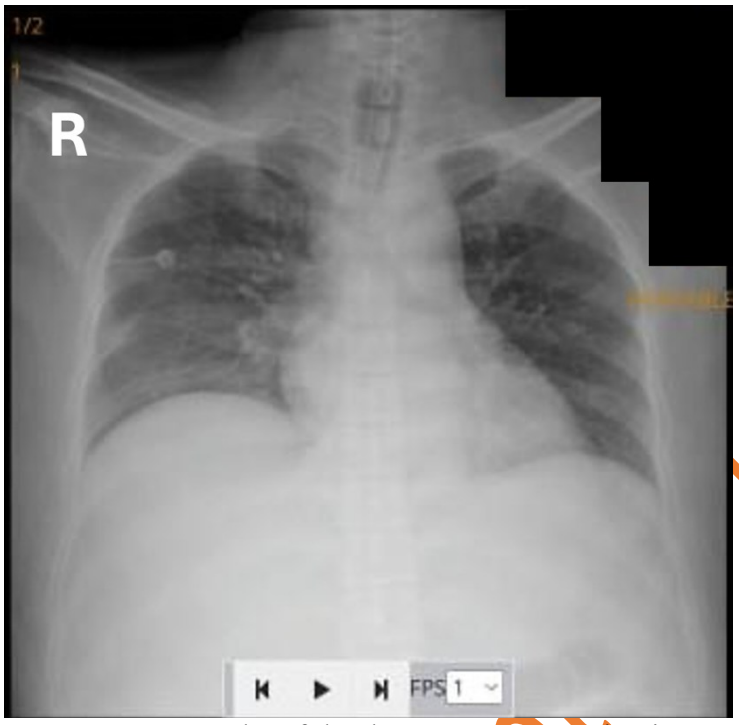

Figure 1. Results of the thorax X-ray examination

The patient was treated for 14 days in the ICU. During treatment, he received IVFD therapy NaCl 0.9% 20 mL/hour, Meropenem iv 2 g/8 hours, Metronidazole iv 500 mg/8 hours, Fluconazole iv 200 mg/24 hours, some continues infusion therapies: Esomeprazole titrated 8mg/hour, Furosemide titrated 5 mg/hour, Fentanyl titrated 30 mcg/hour, Norepinephrine titrated 0.3 mcg/kg/minute, Paracetamol iv 1 g/8 hours, Vitamin K iv 10 mg/8 hours and plasbumin iv 25% extra (Grifols Therapeutics Inc, USA). The patient received continuous renal replacement therapy (CRRT) using continuous veno-venous hemodiafiltration (CVVHDF) modality settings, including a preblood pump of 240 mL, fluid removal per hour of 80 mL/hour, heparinization of 200 IU/hour, a return pressure of 130-121, a transmembrane pressure (TMP) of 60-100, filter pressure drop (FF) of 16%, and an access pressure range of 180-(-115).

During treatment, the patient received early parenteral nutrition therapy in the form of Clinimix (Baxter International, Inc, USA) 83 mL/hour, 0.9% NaCl (Otsuka Indonesia, Inc, Indonesia) 20 mL/hour, Smoflipid (Fresenius Kabi, USA)17 mL/hour and Tutosol (Kalbe Farma Tbk, Inc, Indonesia) 40 mL/hour for the first 4 days of treatment. In the NGT, reddish black, brownish stoma products, and serous hemorrhagic drains were found.

Furthermore, on the 5th day of treatment, the residue from NGT decreased, so additional enteral nutrition was given in the form of a liquid diet through NGT. On the 5th to 7th day, the patient received nutritional therapy in the form of Kabiven (Fresenius Kabi, USA) 40 mL/hour, enteral nutrition, and 0.9% NaCl 30 mL/hour. On the 12th day of treatment, there was an improvement in the patient's condition. The patient's consciousness became compos mentis, blood pressure 110/75 mmHg, heart rate

88 times/minute, SpO_2 98%. The patient was extubated; then, parenteral nutrition therapy was stopped on the 14th day of treatment, and the patient received enteral and oral nutrition. Furthermore, on the 15th day of treatment, the patient is allowed to move to the ward.

Table 2. Follow-up therapy while the patient is in the ICU

Therapy	Day-1	Day-5	Day-7	Day-14
IVFD NaCl 0.9%	20 mL/hours	30 ml/hours	30 ml/hours	20 mL/hours
Meropenem iv 2 g/8 hours	2 g/8 hours	2 g/8 hours	1g/8 hours	1g/8 hours
Metronidazole iv	500 mg/8 hours	500 mg/8 hours	500 mg/8 hours	-
Fluconazole iv	200 mg/24 hours	200 mg/24 hours	-	-
Esomeprazole titrated	8mg/hours	-	-	-
Omeprazole iv	-	40 mg/12 hours	40 mg/24 hours	40 mg/24 hours
Furosemide titrated	5 mg/hours	5 mg/hours	-	-
Fentanyl titrated	30 mcg/hours	30 mcg/hours	30 mcg/hours	-
Norepinephrine titrated	0.3 mcg/kg/minute	0.2 mcg/kg/minute	0.1 mcg/kg/minute	-
Paracetamol iv	1 g/8 hours	1 g/8 hours	1 g/8 hours	1 g/8 hours
Vitamin K iv	10 mg/8 hours	-	-	-
Plasbumin iv 25% (extra)	-	v	-	-
Clinimix	83 mL/hour	-	-	-
Smoflipid	17 mL/hour	-	-	-
Tutosol	40 mL/hour	-	-	-
Kabiven	-	40 mL/hour	40 mL/hour	-
Enteral Nutrition (liquid diet)	-	6x50 mL	6x150 mL	6x250 mL
Oral Nutrition (high calorie, low protein)	-	-	-	Start after extubation
CRRT	-	v	-	-

DISCUSSION

Nutritional therapy, as part of the management of critically ill patients, must be given at the right time, most effectively, and in doses appropriate to individuals to avoid malnutrition during treatment.[9] Sepsis accompanied by increased metabolic stress worsens the nutritional status of patients, especially patients who are already in poor nutritional condition when they are admitted to the ICU. Early enteral nutrition is given to correct micronutrient and vitamin deficiencies, provide adequate protein, and ensure adequate nonprotein caloric intake because patients who are well nourished will produce sufficient endogenous energy. After resuscitation, increased protein and caloric levels are needed to reduce the loss of body mass (LBM) and promote recovery. High protein intake is essential to achieve adequate nutrition and facilitate functional recovery and LBM in sepsis patients.[14]

Appropriate nutritional management is necessary as part of an integrated overall treatment approach since AKI seldom manifests as isolated organ failure in critically sick patients, but rather as a component of multiple organ failure syndrome. Nutritional support and renal replacement therapy (RRT) must be closely integrated for patients with AKI, particularly when extensive RRT is being administered, such as continuous venovenous hemofiltration (CVVH), prolonged daily intermittent dialysis, or sustained low-efficiency dialysis (SLED). Nutritional support in AKI patients is essential, and the typical metabolic abnormalities associated with the acute uremic state and its complications must be considered.[15] In this patient, a diagnosis of decreased consciousness after the Hartmann's procedure, septic shock, AKI, and peptic ulcer was obtained.

Malnutrition worsens the clinical condition of patients with sepsis and results in a longer ICU stay. In conditions where enteral nutrition cannot be given at all, total parenteral nutrition should be started as soon as possible in critical sepsis patients with malnutrition.[14] Currently, ICU practitioners believe that AKI patients in the ICU are at high risk of malnutrition. A study by the International Society of Renal Nutrition and Metabolism (ISRNM) introduced a standard definition related to malnutrition in AKI with the term "protein-energy wasting". This condition indicates a decrease in body protein stores and energy fuels, which can occur in AKI, regardless of the cause, and is associated with decreased functional capacity due to metabolic stress. The diagnosis of protein-energy wasting in AKI uses four categories of diagnostic criteria, namely, biochemical (such as albumin or prealbumin), weight loss, decreased muscle mass, and decreased protein intake. Protein energy wasting can be detected by a decrease in BMI to <20 kg/m2, weight loss >10% in 6 months, serum albumin <35 g/l, and transthyretin (prealbumin) <300 mg/l.[16] In this patient, nutritional status was measured from BMI with a body weight of 64 kg and a height of 167 cm, so the BMI was 22.9 kg/m2 (good nutritional status).

The early acute phase of sepsis is not always in a hypermetabolic state because it does not consistently result in increased caloric needs. In the early phase of sepsis, the total energy expenditure (TEE) to resting energy expenditure (REE) is 1.0–1.1.[14] Nutritional status is one of the main prognostic factors in AKI patients in the ICU. Protein-energy wasting affects outcomes such as length of stay in the ICU and hospital, complication rates, and mortality rates. Severe malnutrition is a predictor of in-hospital mortality regardless of other AKI complications and comorbidities.[15]

Many factors are indications for parenteral nutrition, such as calorie needs cannot be met through enteral nutrition after more than 5 days, enteral nutrition is postponed due to gastrointestinal obstruction, there is a high-output fistula, enteral nutrition cannot be given owing to gastrointestinal bleeding (peptic ulcers), enteral nutrition cannot be given in patients with abdominal compartment syndrome, and there is a high risk of aspiration. In this patient, parenteral nutrition was given based on indications that enteral nutrition cannot be given owing to gastrointestinal bleeding (peptic ulcer).[9]

Early nutritional therapy (within 72 hours) in trauma patients has the advantage of reducing the risk of sepsis by up to three times compared with delaying nutritional administration. Nutrition can be given immediately after hemodynamics are stable. Another study revealed that providing nutrition in the first 48 hours after trauma is associated with decreased infection rates, mortality, and length of hospitalization.[6] European Society for Clinical Nutrition and Metabolism (ESPEN) states that every critically ill patient who is treated for more than 48 hours in the ICU is considered at risk of malnutrition. Therefore, nutritional therapy must be determined by time, method of administration, and calorie/protein targets. Oral nutrition is given if possible; if not, the next choice is enteral nutrition early, which increases gradually over the first 48 hours. If neither is possible, parenteral nutrition can be given.[17]

The recommended time for administering parenteral nutrition is if, within the first 7 days, the patient cannot tolerate enteral nutrition, even though measures have been taken to optimize the administration of enteral nutrition.[9] In these patients, parenteral nutrition is given directly because they are considered at risk of malnutrition for several reasons, such as being critically ill for more than 48 hours, having post-operative gastrointestinal cancer, having a period of fasting post-operatively, and being unable to receive enteral nutrition due to gastrointestinal bleeding (peptic ulcers).

The basis of providing nutritional therapy to trauma patients is preventing acute protein malnutrition and modulating the immune response. In trauma patients, hypercatabolism can cause acute protein malnutrition and can then cause immune system disorders.[18] Patients with moderate trauma require nonprotein calories (NPCs) of approximately 25–30 kcal/kg/day and 1.0–1.5 g/kg/day of protein, with an NPC: nitrogen ratio of 80–120. If stress has been reduced but energy needs remain the same, protein supplementation can be reduced to 1.0–1.2 g/kg/day with an NPC: nitrogen ratio of 130–160. Patients with these conditions are not recommended to receive an immune-enhancing diet. This is due to immune-enhancing diets (2.2–2.5 g protein kg/day) that are higher in L-arginine, glutamine, fatty acids, and/or nucleotides may be beneficial since they result in fewer infections, lower MOF, less antibiotic use, and shorter hospital stays than polymeric diets.[19] This diet is maintained for 7–10 days only and then changed to a standard diet to prevent energy deficit.[6] The protein catabolic rate (PCR) in AKI patients receiving PN or EN nutrition or a combination of both varies from 1.4–1.8 g/kg/day. Patients with AKI require a minimum intake of 0.25 g of nitrogen/day to achieve a balanced or nearly positive nitrogen balance.[15]

AKI patients on RRT should receive no more than 30 kcal of NPCs, or 1.3 times basal energy expenditure (BEE) as determined by the Harris-Benedict equation, and at least 1.5 g/kg/day (= 0.25 g N/kg/day) of protein. To make up for protein and amino acid losses during RRT, particularly when high-flux dialysis or CRRT is used, protein intake should be raised by around 0.2 g/kg/day in this situation. The losses may be computed as around 5 g-10 g of protein per day and 0.2 g of amino acids/l ultrafiltrate (up to 10-15 g of amino acids per day).[15]

About 30–35% of the overall nonprotein energy source should come from lipids. Patients can get parenteral nutrition by receiving 0.8–1.2 g/kg/day of lipid from a 10–30% lipid emulsion or as a component of a three-in-one complete nutrient combination that is sold commercially. When blood triglyceride levels are above 400 mg/dL (~5.3 mmol/L), lipid therapy should be stopped. Lipids should be administered for 18 to 24 hours.[15] This patient was given parenteral nutrition in the form of Clinimix 83 mL/hour, Smoflipid 17 mL/hour, and Tutosol 40 mL/hour from the 1st day to the 4th day in the ICU with a target of 1920 kcal/day and 96 g/day protein. On the 5th to 7th day of treatment, the patient received partial parenteral nutrition, given Kabiven as much as 40 mL/hour with a target of 1800 kcal/day calories and 64 g/day protein. In addition, the patient also received enteral nutrition therapy in the form of a liquid diet of 6 x 50 mL. On the 7th to 14th day of treatment, the patient received Kabiven therapy with the same dose plus a liquid diet in the form of milk as much as 6x150 mL, which was increased gradually every day to a maximum of 6x250 mL. On the 14th day of treatment, after the patient was extubated, parenteral nutrition therapy was stopped, and the patient received oral nutrition (high-calorie, low-protein) and enteral nutrition.

Parenteral nutrition through central venous access is recommended. The peripheral venous route can only be used for short-term parenteral nutrition and low osmolality of nutrient fluids. The infusion set for parenteral nutrition is the same as that for other infusion fluids; the infusion set is replaced every day if it is lipid-based and every 2–3 days if it does not contain lipids. If a multilumen catheter is used, 1 lumen should be used specifically for parenteral nutrition, separate from the administration of drugs and other infusion fluids.[9] In certain conditions where there is no central venous access, parenteral nutritional supplements can be given through peripheral venous access with an osmolality of no more than 900 mOsm/L. This prevents phlebitis, inflammation, and pain.[11] In this patient, parenteral nutrition was administered via central venous access, namely, the left jugular vein.

Stress-related gastrointestinal (GI) mucosal damage, such as peptic ulcers and stress gastritis, is a danger for critically sick patients who are brought to the ICU. Following acute physiologic shocks such as burns, major surgery, sepsis, bleeding, or traumatic damage, these problems may develop quickly. Before the widespread use of antisecretory drugs like proton pump inhibitors (PPIs) and histamine-2 receptor antagonists (H2RAs), surgeons treating critically sick patients conducted effective gastric bleeding surgery.[20] PPIs should be administered intravenously continually until the bleeding is under control. Broad-spectrum antibiotics ought to be administered to individuals exhibiting symptoms of sepsis.[21] This patient was given the PPI Esomeprazole at a dose of 8 mg/hour, the antibiotics Meropenem at 2 g/8 hours and Metronidazole at 500 mg/8 hours. On the 8th day of treatment, there was clinical improvement, so the Meropenem dose was reduced to 1 g/8 hours.

CONCLUSION

Nutritional therapy is an important factor in critically ill patients. Nutritional therapy should be given at the right time, in the most effective way, and in doses that are appropriate for individuals to avoid malnutrition during treatment and

improve patient outcomes. The first route of nutritional therapy is oral; if not possible, the next choice is enteral, and if neither is possible, the choice is parenteral.

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