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Case Report

Medical Nutrition Therapy in Critically Ill Patients with Metabolic Encephalopathy, Diabetes Mellitus, Cerebral Infarction, and Status Epilepticus Complicated by Severe Protein-Energy Malnutrition: Case Report

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ABSTRACT

Glucose homeostasis disturbance is a common complication among patients in intensive care units (ICUs), frequently resulting in stress-induced dysglycemia. Individuals with diabetes mellitus (DM) are particularly susceptible to hyperglycemia and face a higher risk of severe hypoglycemia due to overtreatment. Particularly for patients on insulin or glucose-lowering drugs, it is crucial to maintain regular meal patterns in terms of timing, food type, and quantity. The 63-year-old female patient in this case study was referred from the neurology department after experiencing diminished awareness and going two days without eating. She had experienced multiple seizures lasting more than five minutes and presented with a nasogastric tube (NGT) insertion showing 150 mL of greenish gastric residual. The patient reported reduced intake over the past week due to nausea and headaches, occasional vomiting, intermittent fever, and a weight loss of 2.2 kg (4.8%) within one week. Medical nutrition therapy (MNT) was initiated to ensure adequate nutrient intake through enteral and parenteral routes, followed by a gradual transition to oral feeding. This approach aimed to improve the patient's nutritional and metabolic status through personalized and adequate nutritional care. The patient's clinical condition was managed concurrently, with continuous monitoring of intake, anthropometry, and laboratory parameters to evaluate the intervention's effectiveness. This case highlights that proper medical nutrition therapy for critically ill patients with metabolic encephalopathy, diabetes mellitus, cerebral infarction, and status epilepticus complicated by severe protein-energy malnutrition can lead to significant improvements in clinical outcomes.

Introduction

A common complication in patients in intensive care units (ICUs) is glucose homeostasis disturbance, which leads to stress-induced dysglycemia. Patients with diabetes mellitus (DM) are more susceptible to hyperglycemia and are at a higher risk of excessive glucose correction, which may lead to severe hypoglycemia [1]. A class of metabolic illnesses known as diabetes mellitus (DM) is typified by

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hyperglycemia brought on by deficiencies in either insulin action or secretion, or both. Diabetes-related chronic hyperglycemia is linked to long-term harm, malfunction, and failure of many organs, including the kidneys, heart, blood vessels, nerves, and eyes [2].

Insulin resistance, insufficient insulin secretion, and excessive or incorrect glucagon production all contribute to type 2 diabetes mellitus, which is defined by hyperglycemia. Numerous neuropathy, macrovascular, and microvascular problems are linked to uncontrolled type 2 diabetes. Retinopathy, nephropathy, and perhaps neuropathy are examples of microvascular problems associated with diabetes. Peripheral and coronary artery diseases are examples of macrovascular problems. Both peripheral and autonomic nerves are impacted by diabetic neuropathy [3].

Insulin resistance is the basis of type 2 diabetes, and β-cell failure begins before diabetes develops due to an imbalance between insulin resistance and insulin secretion. De Fronzo stated that β-cell function decreases by approximately 20% at the onset of glucose intolerance. Therefore, it is clear that the treatment approach for type 2 DM should aim to improve insulin resistance and restore β-cell function [4-5]. Nutritional therapy is one of the four pillars in the management of diabetes [5-6].

Meal planning and nutritional management for people with diabetes follow the same guidelines as for the general public, which include eating a balanced diet that satisfies their calorie and nutritional requirements. It is essential for individuals with diabetes to emphasize regularity in meal timing, type, and portion size, especially for those on glucose-lowering medications or insulin. However, this approach differs between critically ill patients and those under outpatient management. This case report discusses the management of a diabetic patient in the critical care setting.

Case Report

A 63-year-old female patient was referred from the Neurology Department with complaints of no oral intake for the past 2 days due to decreased consciousness. Prior to this, the patient experienced seizures more than three times, each lasting more than 5 minutes. A nasogastric tube (NGT) was inserted 1 day ago, with a residual volume of 150 cc, greenish in color. The patient's reduced intake has been ongoing for the past week due to nausea and headaches, with occasional vomiting in the last week. There were no reports of coughing or shortness of breath. The patient had a fluctuating fever, but currently, no fever is present. There was a 2.2 kg (4.8%) weight loss over the past week. Medical nutrition therapy was initiated to ensure nutrient intake through enteral and parenteral routes, which was then gradually combined with oral therapy to improve the patient's nutritional and metabolic status through adequate nutritional care. Additionally, management of the patient's clinical condition was performed. Monitoring and evaluation of intake, anthropometry, and laboratory parameters were carried out to assess the effectiveness of the nutritional intervention. The macronutrients given to the patient included 0.8 g of protein, which was progressively raised to 1.7 g/kg of ideal body weight per day, or 34.24–72.8 grams (12.4–18.2%). Carbohydrates were provided at 40%–45%, corresponding to 110–180 grams, and fats at 47.6%–34%, corresponding to 8.2–65.4 grams. The total energy intake (KET) was started at 30% (1,100 kcal) via enteral feeding and gradually increased until 100% KET (1,600 kcal) was achieved. Nutritional therapy was administered orally once the patient's consciousness improved. Fluid requirements were given at a rate of 1 mL per kcal. Supplementation included zinc 20 mg/24 hours, vitamin B complex 2 tablets every 8 hours, curcuma 400 mg every 8 hours, and fish oil 1,000 mg/24 hours.

Discussion

Metabolic encephalopathy is a global brain dysfunction condition that causes changes in consciousness, behavior, and seizures, resulting from abnormalities within or outside the brain. This condition affects the Ascending Reticular Activating System (ARAS) and/or disrupts its projections to the cerebral cortex, leading to consciousness disturbances and/or seizures. The mechanisms underlying brain dysfunction are multifactorial, including changes in blood flow, neurotransmitter dysfunction, energy metabolism failure, and cellular depolarization [6].

In summary, metabolic encephalopathy is a brain dysfunction with causes originating both intra- and extracerebrally. The processes include metabolic disturbances (such as electrolyte imbalances, serum osmolarity, renal function issues, liver dysfunction), various deficiencies (such as metabolic substrates, thyroid hormones, vitamin B12, etc.), toxins (medications, alcohol, etc.), or systemic toxic abnormalities (e.g., sepsis). Metabolic encephalopathy presents as diffuse brain dysfunction, typically with a rapid onset and fluctuating levels of consciousness (including attention and concentration) [6].

A variety of brain conditions known as metabolic encephalopathy are brought on by systemic illnesses such diabetes, liver disease, renal failure, and heart failure rather than by basic anatomical problems. Usually occurring either abruptly or subacutely, it can be reversed by treating the underlying systemic illness. On the other hand, metabolic encephalopathy may cause subsequent brain structural damage if left untreated. Metabolic encephalopathy comes in two primary forms: one brought on by a lack of oxygen, glucose, or metabolic

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cofactors, which are often obtained via vitamins, and the other brought on by malfunctioning peripheral organs. [6].

There are several different illnesses that might result in chemical imbalances in the brain that are enough to produce encephalopathy and coma. Vitamin deficiencies, hereditary diseases, and certain neuroendocrine disorders at specific stages can disrupt brain metabolism and lead to encephalopathy. Toxic encephalopathy can also result from exposure to other heavy metals and organic solvents. Ethanol, commonly found due to its widespread use, can lead to permanent brain damage when consumed in excess, especially when associated with vitamin deficiency and malnutrition. Clinically, brain damage from prior acute drinking episodes is frequently associated with alcoholic cirrhosis-induced hepatic encephalopathy [6].

Metabolic encephalopathy is characterized by a generalized depression of brain function, including awareness. The neocortex's diminished integrative capacity might be the cause of the influence on awareness. The ascending reticular activating system (ARAS), which is made up of certain brainstem nuclei and their major fiber routes, mediates the activation of the neocortex and other frontal brain areas involved in cognition. Activation of the ARAS ascending pathways relays through the thalamus to the neocortex. Metabolic encephalopathy is caused by chemical changes in the ARAS centers of the neocortex and brainstem [7].

Pupils may look tiny yet reactive, and respiratory function may decline. Asterixis, also referred to as "flapping tremor," is frequently seen when encephalopathy worsens, particularly in cases of liver illness, uremia, and sedative drug overdose. Loss of postural tone in voluntary muscles of the limbs, trunk, head, or tongue causes asterixis. Seizures, such as those caused by hypoglycemia and acute liver failure, and eventually Cheyne-Stokes breathing patterns as a result of the loss of brainstem respiratory control are signs of advanced stages of metabolic encephalopathy. Furthermore, a number of metabolic encephalopathy instances, such as those brought on by vitamin shortages and the use of hazardous substances, are typified by specific metabolic alterations in the cerebellum and basal ganglia that impair coordination and movement control [6].

Severe Protein-Energy Malnutrition

The diagnosis of severe protein-energy malnutrition in this patient is based on the anamnesis, physical examination, and supportive tests, with the use of screening and assessment tools such as the Subjective Global Assessment (SGA), which yielded a score of C. The patient has a history of decreased intake due to nausea and headache, leading to reduced oral intake and weight loss. According to the ASPEN 2012 criteria, malnutrition can be diagnosed by meeting the following criteria: 1) inadequate calorie intake, 2) weight loss, 3) muscle mass reduction, 4) subcutaneous fat loss, 5) localized or generalized fluid accumulation, or a decrease in functional status [7].

ESPEN uses several criteria to define malnutrition: Option 1: BMI < 18.5 kg/m²; Option 2: a low free-fat mass index or a lower BMI combined with an unplanned weight reduction of 10% or more than 5% over three months. In this case, the patient exhibited: 1) Decreased oral intake due to nausea and headaches, 2) Weight loss: the family reported approximately 5% weight loss, 3) Decreased albumin levels (2.8), which serve as a laboratory indicator for malnutrition risk [7].

Adult hospitalized patients' energy needs are usually assessed according to their age, sex, body mass index, body composition, and clinical condition. Most individuals need between 20 and 35 kilocalories per kilogram (kcal/kg), according to the ASPEN standards. The gold standard for determining hospitalized patients' energy needs is indirect calorimetry, which calculates basal metabolic rate by measuring total body oxygen consumption and carbon dioxide exchange [8-9].

Predicting energy expenditure (EE) in critically ill patients is challenging, as predictive equations fail to match measured EE in about 80% of patients, and protein loss cannot be estimated without specific measurements. Many studies report a high incidence of unintentional underfeeding (i.e., actual calorie and protein intake lower than prescribed amounts). The relationship between prescribed calorie intake and various outcome variables has been reported by multiple research groups [10] (Figure 1).

Due to inconsistent findings from prospective trials, the ideal macronutrient intake is still unknown. The absence of precise monitoring instruments contributes to this ambiguity. Both underfeeding and overfeeding may be avoided with the aid of computerized information systems. Additionally crucial is the interaction between protein consumption and energy [10]. Optimizing protein consumption is as complicated as optimizing calorie intake. Nutrient intake, de novo generated amino acids, and products of protein tissue breakdown all affect the pool of free amino acids. These amino acids are either oxidized and removed as urea, integrated into proteins, or engaged in regulatory mechanisms. The quantity of protein required to maintain neutral tissue protein balance, at least in physiological conditions, is known as minimal protein needs [10].

However, the kind of proteins generated varies greatly from healthy settings, and protein breakdown accelerates dramatically during serious disease. Protein synthesis rises in patients with multiple organ failure, according to research by Roo Ackers et al. Furthermore, clearance systems, particularly renal function, are frequently compromised, and a number of pathways that may be

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controlled by amino acids are triggered. Consequently, information gathered from healthy participants cannot be used to determine the ideal protein intake in critically sick patients [10].

Increased proteolysis via the proteasome/ubiquitin pathway is linked to lean body mass loss and decreased physical activity in critically sick individuals. Based on this finding, it is hypothesized that the increased protein requirement is caused by: (a) anabolic resistance, which increases the need for amino acids to achieve the same muscle synthesis rate; (b) acute-phase protein synthesis, which requires amino acids; (c) cysteine, a rate-limiting step in glutathione synthesis, to limit oxidative stress; and (d) preventing glutamine depletion in muscle and plasma [10].

The construction, upkeep, and development of cells, tissues, enzymes, hormones, antibodies, and transportation all depend on protein. The patient's age, weight, nutritional state, and degree of metabolic stress from illness or injury all influence how much protein they need each day. For healthy people, 0.8 g/kg of protein is the Dietary Reference Intake (DRI). Acute or chronic illnesses frequently cause rapid muscle catabolism and negative nitrogen balance in hospitalized individuals. In average, adults under metabolic stress need 1.2–2.0 g/kg of protein [8–9].

According to recent data, one research found inconsistent results, while another found that a high protein diet (1.2 to 1.5 g/kg per day) was linked to improved outcomes (Figure 2). According to Ishibashi et al., the most detrimental total body protein balance was linked to 1.5 g/kg daily. Total body or tissue protein balance, circulating protein or amino acid levels, physiological processes (muscle strength, immunological competence, insulin sensitivity, glutathione levels, and oxidative stress), and finally clinical consequences are biomarkers for the best possible intake of protein and amino acids. Techniques for assessing lean tissue mass using ultrasound or CT scans may help adjust protein intake more accurately, although further research is needed [10].

To prevent shortages in critical fatty acids, lipids should be included in nutritional support. Additionally, fat-soluble vitamins and components that are involved in receptor activation, eicosanoid synthesis control, cell membrane structure and function, and gene expression modification in metabolic pathways and inflammatory mediators are provided by lipid formulations. An inflammatory state and a number of metabolic abnormalities, such as hypertriglyceridemia, elevated free fatty acids, and decreased cholesterol and LDL/HDL lipoproteins as a result of decreased lecithin-cholesterol-acyltransferase activity, are commonly observed in critical illness [11].

Additional frequent observations include decreased carnitine levels, increased lipolysis, and decreased enteral fat absorption, which change mitochondrial activity and fatty acid oxidation. Inflammatory processes can be influenced and modified by the kind of lipid that is supplied in nutritional-metabolic support. Lipid type is a crucial element since omega-3 fatty acids in fish oil have anti-inflammatory properties, omega-9 fatty acids in olive oil have a neutral impact, and omega-6 fatty acids in soybean and sunflower oils have pro-inflammatory properties [11].

Critically sick patients may benefit from fatty acid blends that lessen the pro-inflammatory effects of omega-6 fatty acids included in conventional emulsions. Formulations of different oils, such as olive oil, fish oil, medium-chain triglycerides, soybean oil, and sunflower oil, are used as strategies to decrease omega-6 fatty acids. Results from published research to date have been inconsistent [11].

Although further study is required to validate these findings, a meta-analysis by Manzanares et al. found that methods to reduce omega-6 fatty acids might lower mortality, the number of days on mechanical ventilation, and the length of stay in an intensive care unit. Grau-Carmona et al. looked into how omega-3 fatty acids affected the incidence of nosocomial infections and other clinical outcomes in critically sick surgical patients in later research, and they found that there was a notable decrease in nosocomial infections. Mortality, days spent on mechanical ventilation, and hospital duration of stay, however, did not show any appreciable variations [11].

Due to patient heterogeneity and the variety of mixtures studied, no general recommendations for critically ill patients can be made. However, it is clear that fish oil-enriched emulsions increase the omega-6/omega-3 ratio, which is likely to have anti-inflammatory effects. According to expert guidelines, a lipid dosage of 0.7–1.3 g/kg/day, or 25%–40% of caloric intake, should be followed, with modifications made if plasma triglycerides above 400 mg/dl (Figure 4) [11]. In this patient, energy, protein, carbohydrate, and fat intake were monitored as follows:

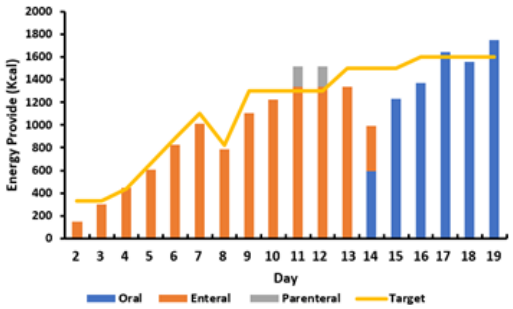

Figure 1- Energy Intake Monitoring

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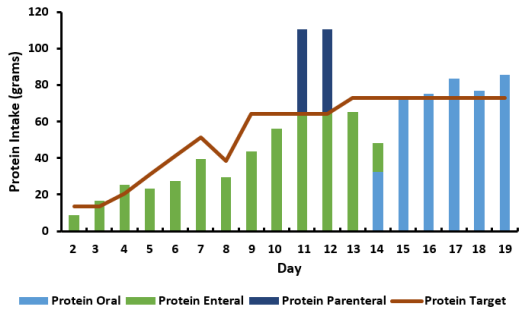

Figure 2- Protein Intake Monitoring

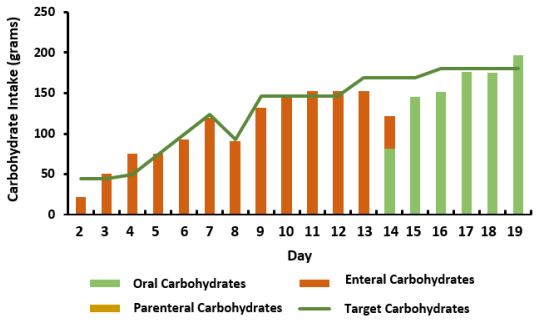

Figure 3- Carbohydrate Intake Monitoring

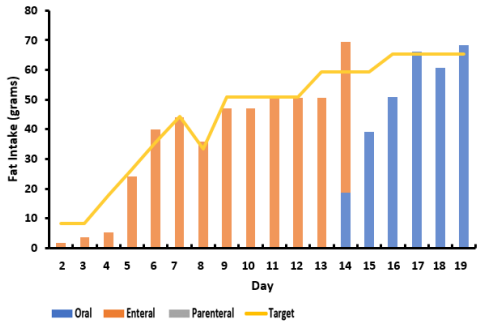

Figure 4- Fat Intake Monitoring

In nutritional evaluations, micronutrients are frequently disregarded, which can lead to less than ideal nutrition supply for intensive care unit patients. Zinc, selenium, copper, and vitamins C, E, D, and B are examples of micronutrients that are engaged in a variety of metabolic processes. They can facilitate a variety of enzyme functions or act as catalysts. The severity of the underlying disease, pre-existing malnutrition, and the side effects of treatment plans or procedures can all lead to micronutrient deficiencies. Micronutrients are redistributed from the circulation to organs implicated in acute-phase synthetic processes as a result of subsequent inflammatory reactions [10].

Patients are provided with enteral nutrition support and supplementation. Once the patient is stable, enteral feeding is initiated. Enteral feeding must be started as soon as possible, and physiological evidence supports this. The majority of critically ill patients benefit from the non-nutritional benefits of enteral nutrition during the first week of intensive care unit (ICU) admission. These effects include protecting gut integrity, boosting immunoglobulin A (IgA) production, supporting microbiome diversity, and conserving gut-associated lymphoid tissue (GALT). By maintaining mucosa-associated lymphoid tissue (MALT), promoting anti-inflammatory Th2 lymphocytes, and activating regulatory T cells (Tregs), enteral nutrition also contributes to the immune response [12].

Additional metabolic advantages of enteral feeding include decreased production of advanced glycation end products (AGEs) and increased incretin secretion. Early enteral feeding is linked to lower mortality and infection rates than delayed enteral nutrition, according to a meta-analysis of randomized controlled studies. Nevertheless, there was insufficient statistical power and heterogeneity in the included clinical studies. Furthermore, the term "early nutrition" is still ambiguous, with different interventional studies using definitions that range from three to seven days [12].

Early enteral nutrition versus early parenteral nutrition over the first five days in a critically sick population were studied in the CALORIES experiment, a randomized controlled trial. There were no differences in short-term results, and the majority of patients in both groups failed to reach their energy expenditure goals (Figure 3). Enteral vs parenteral feeding had no influence on overall mortality, according to a recent meta-analysis that incorporated the findings of the CALORIES study. Enteral feeding, however, was linked to shorter ICU stays and fewer infection problems [13].

Dysglycemia in Patients with Metabolic Encephalopathy

A major physiological stressor, encephalopathy causes the hypothalamic-pituitary-adrenal axis and sympathetic nervous system to become active, which raises catecholamine levels. This response is part of the body's acute adaptation mechanism, often accompanied by transient hyperglycaemia. While mild stress-induced hyperglycaemia can serve a protective role, excessive hyperglycaemia during severe illness becomes pathological and may necessitate medical intervention to prevent further complications [1,14].

Instead than being a direct cause of poor outcomes, hyperglycemia is often viewed as a sign of the severity of the disease in critically sick patients. The hyperglycaemic effects of catecholamines frequently cause acute hyperglycemia, which is defined as a mismatch between the body's elevated glucose demand and insufficient insulin production. In contrast, chronic illness with substantial tissue damage can induce insulin resistance, further exacerbating glycaemic imbalance [1,14].

Patients with pre-existing diabetes mellitus are more vulnerable to pronounced stress-induced

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hyperglycaemia. This is mostly caused by hyperglucagonemia and insulin resistance in type 2 diabetes or insulin shortage in type 1 diabetes. Consequently, diabetic patients often require tighter glycaemic control and pharmacologic interventions such as insulin to mitigate adverse metabolic effects during acute illness [1,14-15]. Controversy remains regarding the ideal approach to glycaemic control in critical care. The Leuven trials demonstrated improved patient outcomes with intensive glucose control, while subsequent studies, including the NICE-SUGAR trial, found increased mortality with tight control. These conflicting findings have prompted debate over whether benefits were derived from glucose control itself or from factors such as monitoring accuracy and the supportive environment in the ICU [16-17].

Pre-admission glycaemic status has emerged as an important determinant of patient outcomes. During intensive care unit stays, elevated HbA1c levels have been linked to a higher risk of hypoglycemia and death. Although randomized trials have produced mixed outcomes, current clinical practice considers the avoidance of both hyperglycaemia and hypoglycaemia as essential components of neuroprotective care, particularly in patients with neurological involvement such as encephalopathy [17].

Nutrition management in the ICU is closely linked to infection risk and overall outcomes. Some studies suggest that caloric deficits contribute to increased nosocomial infections, while others report no significant effect. Trials like TICACOS and the study by Heidegger et al. indicate that supplemental parenteral nutrition may help close the energy gap and reduce infection risk, but may also prolong ICU stay and increase complication rates [18-19]. Feeding strategies such as norm caloric versus hypocaloric nutrition are influenced by gastrointestinal tolerance and concurrent therapies. While norm caloric feeding is often preferred, it has been associated with higher rates of diarrhoea, especially when combined with antibiotics. In patients with limited tolerance for enteral nutrition, supplemental or total parenteral nutrition may be necessary, though early initiation has also been linked to increased infection rates in some studies [18].

A diabetic diet and a target blood glucose range of 140–180 mg/dL were used to treat the patient in the instance that was described, in accordance with standard critical care guidelines for diabetic patients. Insulin therapy was initiated under endocrinology consultation, beginning with continuous intravenous infusion followed by subcutaneous administration. This approach aimed to stabilize blood glucose levels while minimizing the risks of hypoglycaemia and glucose variability during the acute phase of metabolic encephalopathy [18].

Status Epilepticus in Diabetic Patients with Metabolic Encephalopathy

Although the exact etiology of seizures in diabetes individuals is unknown, physiological variables such oxidative stress, immune system dysfunction, cerebral microvascular damage, disruption of the blood-brain barrier (BBB), metabolic abnormalities, and genetic alterations are thought to be involved. epileptic is characterized by the existence of an epileptic syndrome, a single seizure with a high chance (≥60%) of recurrence, or two or more unprovoked or reflex seizures that occur more than twenty-four hours apart. Certain stimuli, such as visual, auditory, or somatosensory triggers, can cause reflex seizures [20–21].

A medical emergency known as status epilepticus (SE) is brought on by either initiation mechanisms that result in extended seizures or by failing to stop seizures. It can cause serious consequences such as neuronal injury, behavioral changes, and increased mortality, especially when lasting over 30 minutes. Imaging tests like CT or MRI should be used to support the clinical diagnosis of SE in order to identify structural abnormalities, particularly in youngsters, and may include sedation. Blood counts, serum electrolytes, renal function tests, bedside blood glucose, and cerebrospinal fluid studies should all be included of laboratory examinations. It is also essential to assess the use of prior antiepileptic medications [22-25].

Because of the disproportionately high energy and oxygen requirements of the human brain in relation to its size, maintaining normoxia requires careful control of oxygen metabolism and cerebral blood flow. When cerebral blood flow is disrupted, hypoxia, insufficient ATP synthesis, and compromised synaptic plasticity frequently result. Seizures have been shown to cause fluctuations in brain oxygenation, with postictal hypoperfusion and hypoxia linked to acute behavioral deficits and cognitive dysfunction, as seen in both animal models and human studies [26].

Interestingly, while hypoxia has been found to reduce seizure duration in kainic acid-induced SE models, it is also associated with greater hippocampal neuronal toxicity. Studies have revealed a synergistic effect of hypoxemia and hypercapnia in shortening seizures. Wasterlain et al. demonstrated that mechanical ventilation to correct hypoxemia could reduce mortality, though neuronal injury may still occur due to SE itself. In the presented case, the patient was managed with diazepam and phenytoin administered by the neurology team [27-29].

Cerebral Infarction in Diabetic Patients

Due to cerebral ischemia, which can result from an ischemic stroke or be a consequence of cardiovascular illness, patients with diabetes mellitus (DM) are more likely to experience a cerebral infarction. Diabetes is

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linked to several kinds of ischemic stroke, such as lacunar infarcts, major artery occlusions, and thromboembolic stroke, and it also causes atherosclerotic alterations in the heart and cerebral arteries. Through a number of detrimental mechanisms, including oxidative stress, increased blood-brain barrier (BBB) permeability, and activation of inflammatory pathways involving pro-inflammatory cytokines (including TNF-α, IL-1, IL-6, and IFN-γ), macrophages, T cells, and other immune cells, both hypoglycemia and hyperglycemia can worsen brain injury [30].

During ischemia, the lack of oxygen and glucose triggers tissue damage through multiple pathways, including bioenergetic failure, glutamate excitotoxicity, BBB disruption, and oxidative stress, ultimately leading to cell death by necrosis or apoptosis. In diabetic patients, hyperglycemia worsens the injury by promoting inflammation and endothelial damage, leading to leukocyte infiltration into the central nervous system. Therefore, controlling blood glucose levels is essential to prevent the progression of ischemic injury. Additionally, citicoline can be administered as a neuroprotective and neurorepair agent to support brain tissue recovery following ischemic infarction [30].

Leukocytosis and Increased NLR

Normal leukocyte levels range from 5,000–9,000 cells/mm³. When the count exceeds 12,000, it is referred to as leukocytosis. Leukocytes, produced in the bone marrow and lymphatic tissues, play a key role in the body's immune defense. They are classified into granulocytes (neutrophils, eosinophils, and basophils) and non-granulocytes (monocytes and lymphocytes). Neutrophils make up 50–70% of total leukocytes and typically live for 4–5 days [31-32].

In this case, the patient had a leukocyte count >10,000. Elevated leukocyte levels can be triggered by advanced glycation end-products, oxidative stress, and angiotensin II—all consequences of hyperglycemia. These factors stimulate the production of pro-inflammatory cytokines like TNF-α and IL-1β, which contribute to chronic diabetic complications. Higher leukocyte counts are associated with both macrovascular and microvascular complications of diabetes. In this patient, the elevated leukocyte count served as a relevant indicator of type 2 diabetes complications (Figure 5) [32].

Uncontrolled hyperglycemia is associated with impaired innate and adaptive immune responses, making individuals more susceptible to bacterial infections. Chronic complications of diabetes, such as sensorimotor and autonomic neuropathy and peripheral vascular disease, often lead to skin ulcers that are vulnerable to secondary bacterial infections. Moreover, diabetes significantly increases infection-related mortality. Inflammatory or infectious responses in the body typically cause an increase in neutrophils (neutrophilia), which play a crucial role in combating invading pathogens and regulating allergic reactions. Basophils, which release histamine and heparin during inflammation, also contribute to the immune defense. White blood cells (leukocytes) are classified into agranular and granular types based on their appearance and content [32].

Raised C-reactive protein (CRP), hypoalbuminemia, raised cytokine levels, and elevated white blood cells and their subtypes are all indicators of systemic inflammation in the blood. The neutrophil-to-lymphocyte ratio (NLR), which is commonly used to evaluate inflammatory state, is one easy-to-use indicator. NLR serves as an indirect indicator of the balance between the inflammatory activity of neutrophils and the regulatory role of lymphocytes. It has gained recognition as a valuable biomarker to evaluate inflammation and predict outcomes in diseases like cancer, cardiovascular conditions, and infections. Its ease of calculation from routine blood tests has made it a common tool in both research and clinical settings [33-34].

NLR is now generally acknowledged as a trustworthy indicator of immunological response in both infectious and non-infectious circumstances. Innate immunity (neutrophils) and adaptive immunity (lymphocytes) interact dynamically in this system, which is impacted by a number of variables such as age, medication usage, chronic illnesses, stress, and more. Normal NLR ranges between 1–2, with values above 3.0 or below 0.7 considered abnormal. Levels between 2.3–3.0 may signal early pathological processes such as cancer, inflammation, or stress. NLR is highly sensitive to conditions like infection, inflammation, and sepsis and has proven prognostic value. Clinical evidence supports daily monitoring of NLR, especially in critically ill patients, as values above 17 or even 30 may indicate severe illness or systemic inflammation [35-37]. Clinical improvement in sepsis, critical illness, and reduced mortality risk are associated with a decrease in the neutrophil-to-lymphocyte ratio (NLR) below 7. Despite its great sensitivity and limited specificity, NLR is an easy, affordable, and rapid indicator of stress and inflammation that aids in differentiating between severe and moderate illness (Figure 6).

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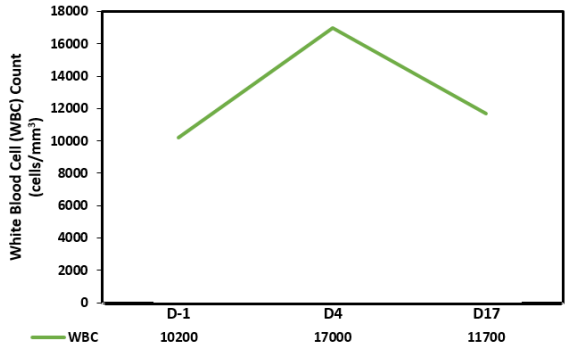

Figure 5- Leukocyte Monitoring

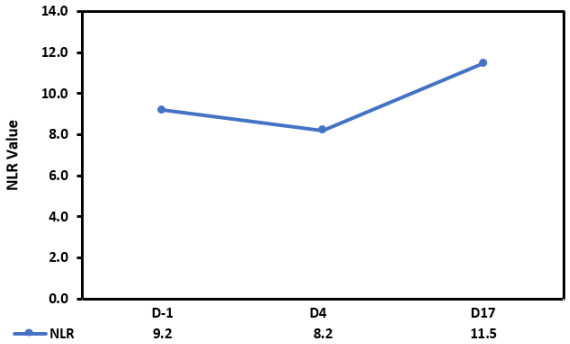

Figure 6- Changes in NLR (Neutrophil-to-Lymphocyte Ratio)

It is advised for regular usage in critical care units and emergency rooms as well as in a number of medical specialties, such as orthopedics, cardiology, neurology, psychiatry, oncology, and surgery. NLR is a possible early warning indicator of underlying pathogenic processes because dynamic changes in it frequently occur hours before clinical symptoms appear. Patient care also involves adequate intake of macro- and micronutrients, particularly zinc, vitamin B complex, and curcuma. Zinc is a key micronutrient for immune function, serving as a catalytic and structural cofactor in various metabolic processes. Similarly, selenium, like zinc, plays essential structural, functional, and enzymatic roles in protein metabolism [38].

Immune System Depletion

The patient experienced immune system depletion, with a Total Lymphocyte Count (TLC) initially recorded at 314/µL, later improving to 1,680/µL before dropping again to the 800s (Figure 6). Management included adequate intake of macro- and micronutrients, specifically zinc, vitamin B complex, vitamin C, and curcuma. Zinc is a crucial micronutrient for immune function, acting as a catalytic and structural cofactor in various metabolic processes. Selenium, like zinc, plays essential structural, functional, and enzymatic roles in protein metabolism. Organ dysfunction in critically sick patients is routinely evaluated, tracked, and treated (Figure 7). Evidence shows that critical illness induces substantial immune system alterations in most ICU patients. Increased morbidity and death are caused by the prolonged anti-inflammatory phenotype (like sepsis-associated immunosuppression) and hyperinflammatory states that these individuals frequently display. Immune suppression is frequently seen in critically sick individuals due to apoptosis-induced lymphopenia and compromised lymphocyte activity. Common inflammatory indicators such as leukocyte count analysis, procalcitonin, and C-reactive protein (CRP) can be used to evaluate functional immunological alterations (Figure 8) [39].

Hyponatremia

Sodium (Na^+) is the primary extracellular cation that plays a crucial role in regulating the action potential of skeletal muscles, nerves, and the myocardium. It also maintains acid-base balance and extracellular fluid (ECF) volume through osmotic pressure regulation. Normal serum sodium concentration ranges between 135–145 mEq/L [40-41]. Imbalances in sodium and water often occur simultaneously and affect serum osmolality. The main factors influencing sodium excretion are glomerular filtration rate and aldosterone levels.

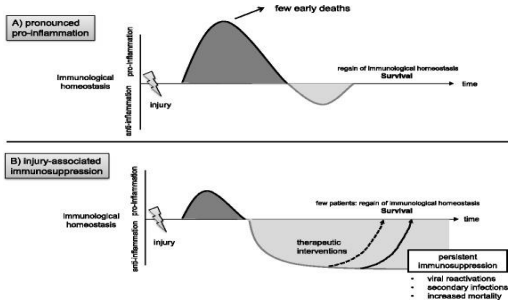

Figure 7- Patterns of injury-related responses among individuals in critically ill patients

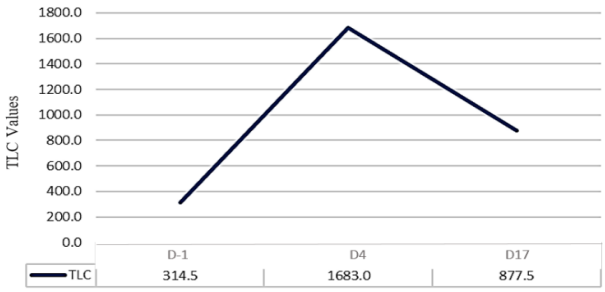

Figure 8- Changes in TLC (Total Lymphocyte Count)

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Serum sodium levels below 135 mEq/L are known as hyponatremia, and they can be caused by either excessive water retention in the ECF, sodium loss from the ECF, or a combination of the two. Factors include the use of diuretics, gastrointestinal fluid loss, fever, open wounds, extensive drainage, and the treatment of sodium bicarbonate for metabolic acidosis put critically sick patients—especially those in the intensive care unit—at higher risk [42-44].

Hyponatremia reflects a relative excess of water due to impaired renal excretion of free water. Causes include the use of loop diuretics, thiazides, osmotic diuretics, and tubular-interstitial disorders that reduce sodium and chloride reabsorption. Non-osmotic triggers of vasopressin release, such as pain, nausea, medications, and hypovolemia, further promote water reabsorption in the renal collecting ducts. Inappropriate administration of hypotonic fluids also contributes to hyponatremia, which may occur in hypovolemic, euvolemic, or hypervolemic states. Cellular swelling, or edema, results from a transfer of water from the ECF into the ICF brought on by a decline in serum sodium. Acute cerebral edema is the most feared consequence of severe hyponatremia (Na^+ <110 mEq/L), which can cause convulsions and coma. In the discussed case, the patient had mild hyponatremia, which was successfully managed with isotonic fluid and oral sodium supplementation (Figure 9) [45-46].

Hypokalemia

A blood potassium concentration below 3.5 mEq/L is known as hypokalemia, and it can be caused by either a shift of potassium into cells or a decrease in the total potassium in the body. While external potassium levels are kept at 4–5 mEq/L, normal intracellular potassium levels fall between 100 and 150 mEq/L. Despite comprising only 2–10% of total body potassium, even small changes in extracellular potassium can have severe consequences, particularly cardiac arrhythmias when levels fall below 3.5 mEq/L or exceed 5.5 mEq/L. Moderate hypokalemia is categorized as 2.5–3 mEq/L, while severe hypokalemia is defined as <2.5 mEq/L and may become life-threatening at <2 mEq/L. Though hypokalemia is associated with increased morbidity and mortality-especially due to cardiac complications its direct role is often masked by coexisting conditions such as hypoglycemia [47-48]. Clinical presentation varies, with many patients remaining asymptomatic, particularly in mild cases. When symptoms are present, they typically relate to underlying causes rather than the potassium deficit itself. Common symptoms include fatigue and muscle weakness, which may present as shortness of breath, constipation, abdominal bloating, or reduced exercise tolerance. In rare cases, muscle weakness may progress to paralysis. Severe hypokalemia, or total body potassium depletion, may lead to rhabdomyolysis, manifesting as muscle cramps and pain. Physical findings are generally normal, although some may present with tachycardia, irregular heartbeat, or tachypnea due to respiratory muscle involvement. Elevated blood pressure may suggest conditions like hyperaldosteronism or rare genetic syndromes, while low blood pressure may point to excessive diuretic or laxative use, bulimia, or tubulopathies such as Bartter or Gitelman syndrome [49].

Treatment involves potassium replacement, though there is no fixed formula for calculating the exact amount required. For moderate to severe hypokalemia, 40–100 mmol of potassium is typically supplemented. In mild cases (3–3.5 mEq/L), oral KCl at 20 mmol/day combined with potassium-rich dietary intake is recommended, although oral supplementation may cause gastric irritation. Approximately 200–400 mEq of potassium is needed to raise serum levels by 1 mEq/L, although this estimate may not always be accurate due to variations in individual body composition and contributing factors. Foods rich in potassium can provide up to 60 mmol/day and are considered beneficial [49]. For patients with mild to moderate hypokalemia and minimal symptoms, oral potassium therapy is usually sufficient. If symptoms such as cardiac arrhythmias occur, or if serum potassium drops below 2.5 mEq/L, more aggressive intravenous therapy is required (Figure 10).

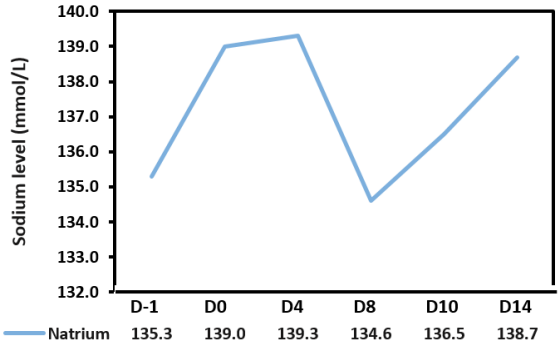

Figure 9- Changes in sodium levels

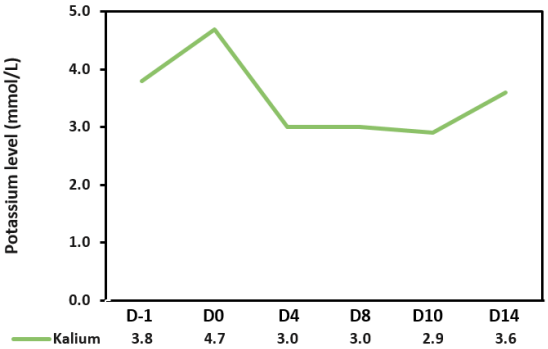

Figure 10- Hypokalemia Monitoring

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Such patients should be closely monitored with continuous ECG and serial potassium measurements. Due to the risk of cardiac complications, many hospitals limit the amount of potassium that can be administered per hour. Hospitalization or emergency observation is often necessary, as replacement may take several hours or more.

Dietary and lifestyle modifications are also essential, especially for patients prone to excessive potassium loss, such as those taking diuretics or laxatives, or those with underlying conditions like digoxin therapy. A potassium-rich, low-sodium diet is generally appropriate, and avoiding potassium-wasting substances like licorice is important in high-risk individuals. In most cases, unless there is severe underlying cardiac disease, activity restrictions are not required (Figure 9) [49].

The patient experienced mild to moderate hypokalemia during the course of treatment. This condition may have been influenced by the patient's glycemic status, which can affect potassium balance. Managing the patient's glycemic condition, ensuring adequate nutritional intake, and administering oral potassium supplements proved effective in restoring the patient's electrolyte balance [49].

Hypoalbuminemia

Albumin is a water-soluble globular protein, distinct among plasma proteins due to its lack of glycosylation. It serves as a major component of plasma protein, accounting for 50–60%, and is synthesized exclusively in the liver at a rate of 12–25 grams per day. Serum albumin levels typically fall between 3.5 and 4.5 g/dL. Transport-related proteins such as serum albumin, alpha-fetoprotein, vitamin D-binding protein, and afamin are also found in albumin. It is essential for the transportation of different chemicals in the blood and for the maintenance of oncotic pressure [50].

Hypoalbuminemia may result from inadequate synthesis (due to malnutrition, burns, infections, or major surgery), excessive breakdown (as in burns or pancreatitis), increased loss through hemorrhage or kidneys, or redistribution into interstitial spaces during inflammatory or surgical conditions. Although albumin infusion offers temporary benefits, correcting underlying protein or amino acid deficiencies is more effective. In some cases of critical illness or malignancy, albumin levels remain low despite nutritional support, and albumin replacement therapy may be needed [51].

Critically ill patients frequently experience hypoalbuminemia, mainly due to blood loss, gastrointestinal losses, increased capillary permeability, and fluid dilution from intravenous therapy. Additionally, elderly patients may already have low albumin levels due to poor nutrition or impaired liver function. Regardless of the cause, hypoalbuminemia is consistently associated with worse clinical outcomes, including higher complication rates and lower short-term survival (Figure 11) [51].

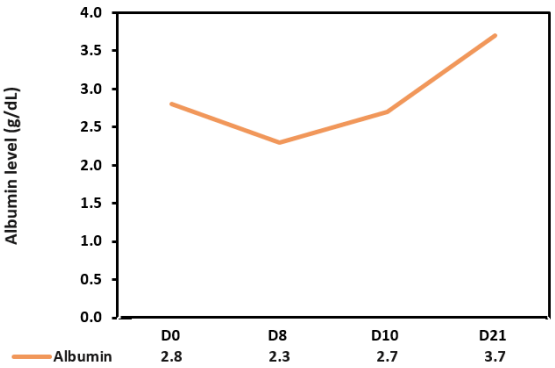

Figure 11- Hypoalbuminemia Monitoring

Decline in Functional Capacity

Handgrip strength (HGS) is clinically used in rehabilitation settings and has been recommended as a fundamental measure for assessing muscle and bone function, as well as identifying weakness and disability. It is essential for assessing general functional state. Since manual dynamometry is a rapid, easy, inexpensive, and non-invasive examination, HGS is regarded as a trustworthy gauge of overall health. Age, sex, anthropometric measurements (height, weight, hand size, and arm circumference), and hand dominance are some of the characteristics that affect HGS. HGS evaluation also contributes to prognosis assessment in both clinical and surgical care, as well as functional evaluations.

In this case, the patient received nutritional therapy involving the provision of macro- and micronutrients to maintain and improve nutritional status. Alongside targeted energy and protein intake, handgrip strength assessments were conducted to evaluate functional capacity. An increase in the patient's handgrip strength was observed, indicating an improvement in functional capacity. In addition to HGS, this improvement was also supported by clinical assessments of the patient's functional status.

Nutrition Knowledge

The patient's and their family's nutrition knowledge was found to be adequate, as revealed during the anamnesis. The patient's family understood the importance of high-protein intake and had proactively prepared additional oral nutritional supplements (ONS) beyond those provided by the hospital's Nutrition Department. For burn patients, it is essential to provide education on the importance of fulfilling macro- and micronutrient requirements in the recommended composition and dosage. The goal of nutrition education for both the patient and their family is to improve their nutritional knowledge, enabling them to understand the objectives and rationale behind the nutritional therapy

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provided. This understanding can support improvements in the patient's nutritional status, enhance immune function, and accelerate the healing process. General nutritional education provided to the patient and family included an explanation of the disease progression and prognosis; motivation to adhere to the recommended dietary intake and meal schedule, which consisted of three main meals per day adjusted to the patient's tolerance, along with three additional snacks; advice on consuming high biological value protein sources and encouragement to consume meals high in calories and protein in line with daily objectives to satisfy energy and protein demands. The patient was also educated on recommended protein-rich food sources to support optimal recovery.

Conclusion

Appropriate medical nutrition therapy in patients with metabolic encephalopathy, diabetes mellitus in critical illness, cerebral infarction, and status epilepticus complicated by severe protein-energy malnutrition can significantly improve clinical outcomes. Adequate provision of energy, protein, and essential micronutrients contributes to the enhancement of functional capacity, reduction of inflammation, and improvement of immune function. Continuous nutritional monitoring and education are essential to support long-term nutritional status and improve the overall quality of life for patients.

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