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Nursing Care of One Patient with Recurrent Hypoglycemia Complicated by Upper Gastrointestinal Bleeding and Mitochondrial Encephalomyopathy

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Abstract: Patients with upper gastrointestinal bleeding (UGIB) face elevated risks of hypoglycemia during fasting periods. If concurrent mitochondrial encephalomyopathy (ME) is present, impaired energy metabolism drastically amplifies such risks. This paper reports a clinical case of a 42-year-old male patient with a 5-year history of ME, admitted for bleeding caused by duodenal bulbar ulcer requiring absolute fasting. Conventional intravenous glucose supplementation failed to stop recurrent refractory hypoglycemia. Based on the pathological mechanism of impaired gluconeogenesis and defective energy utilization resulting from mitochondrial dysfunction in ME patients, the medical team developed three innovative core nursing interventions:(1) Advance the hypoglycemia warning threshold: raise the critical blood glucose cutoff from 3.9 mmol/L to 5.6 mmol/L and implement predictive intervention;(2) Maintain continuous intravenous fluid infusion to supply stable exogenous energy substrates;(3) Administer sequential short-peptide enteral nutrition to build multiple energy supply pathways. This combined protocol successfully stabilized the patient's blood glucose levels, without severe complications, and the patient was discharged smoothly. The intervention strategy breaks through the limitations of single intravenous glucose supplementation and embodies individualized nursing guided by pathophysiological mechanisms. This clinical case provides new insights into blood glucose management for acutely fasting patients complicated with rare metabolic disorders. It reminds clinical nurses to stay alert to special underlying causes of non-diabetic hypoglycemia and advocates comprehensive intervention under multidisciplinary collaboration.

1. Introduction

Upper gastrointestinal bleeding (UGIB) is a common acute clinical disorder with high incidence and mortality rates [1]. Its prevalence varies widely across regions; a study conducted in Uganda reported a prevalence of 24.5% [2]. While most UGIB episodes resolve spontaneously, roughly 20% of patients develop persistent severe hemorrhage, accompanied by rebleeding rates ranging from 10% to 20% and significantly increased mortality risks [1]. Deaths related to UGIB mostly stem from

complications such as multiple organ failure rather than simple blood loss, which greatly increases the complexity of clinical nursing work [3]. Hypoglycemia is a frequent complication in UGIB patients; severe hypoglycemia may lead to irreversible neurological damage or even death, posing major challenges for clinical nurses.

Fasting and stress triggered by UGIB further raise hypoglycemia risks, particularly in patients with pre-existing special underlying diseases. Mitochondrial encephalomyopathy (ME) is an inherited mitochondrial disorder characterized by core pathological changes in mitochondrial-mediated energy metabolism dysfunction, which impairs ATP production in high-energy-consuming tissues including the brain and skeletal muscles [4,5]. This innate defect in energy metabolism severely weakens patients' compensatory capacity for endogenous gluconeogenesis and glycogenolysis under stressors such as fasting or infection, making them highly susceptible to hypoglycemia. Phosphorus-31 magnetic resonance spectroscopy reveals elevated ratios of inorganic phosphate to phosphocreatine and reduced ratios of ATP to phosphocreatine, confirming the fragile energy reserves of such patients [6,7]. However, existing nursing literature lacks systematic nursing protocols targeting ME patients, and the clinical efficacy of routine hypoglycemia prevention and treatment strategies remains unclear for this population.

Against this backdrop, this paper presents a representative complex clinical case: a middle-aged male patient with a 5-year ME history, hospitalized for UGIB caused by duodenal bulbar ulcer requiring absolute fasting. During routine fasting management, the patient suffered repeated refractory hypoglycemia with poor response to conventional glucose elevation therapies. The primary nursing dilemma of this case lay in balancing hemorrhage control and energy supply during the mandatory acute fasting phase, while addressing ME-specific mitochondrial energy metabolism defects. Breaking from standard clinical workflows, the medical team shifted hypoglycemia management from post-event correction to pre-emptive prevention, adopting an individualized blood glucose management regimen featuring an advanced hypoglycemia warning threshold. The patient's blood glucose levels were ultimately stabilized, ensuring smooth progression of clinical treatment.

2. Case Presentation

2.1 General Clinical Data

A 42-year-old male patient was admitted on December 5, 2025, with a primary diagnosis of upper gastrointestinal bleeding, presenting with melena for three days and dizziness plus fatigue for one day. His past medical history included mitochondrial encephalomyopathy and epilepsy.

Admission physical examination findings: body temperature 36.5 ℃, pulse 108 beats per minute, respiratory rate 20 breaths per minute, blood pressure 102/64 mmHg (1 mmHg = 0.133 kPa). The patient appeared lethargic with pale complexion, bilateral hearing loss, grade IV muscle strength in the left limb and grade V muscle strength in the right limb. Emergency fingertip blood glucose measured 3.2 mmol/L.

Laboratory test results: hemoglobin 82 g/L, hematocrit 25.6%, blood lactic acid 4.8 mmol/L, creatine kinase 218 U/L, and normal coagulation function. Gastroscopy identified a 1.2 cm × 1.0 cm ulcer on the anterior wall of the duodenal bulb, Forrest stage IIc (black blood scab visible at the ulcer base without active oozing).

Admission diagnoses:

1) Duodenal bulbar ulcer with hemorrhage;
2) Mitochondrial encephalomyopathy;
3) Moderate anemia.

2.2 Clinical Treatment, Nursing Interventions and Clinical Outcome

Upon admission, the patient received absolute fasting, acid suppression therapy (40 mg esomeprazole intravenously twice daily), fluid resuscitation, intravenous glucose support, and transfusion of 2 units of suspended red blood cells. During hospitalization, recurrent hypoglycemic episodes occurred, with the lowest recorded blood glucose value at 2.1 mmol/L. Most hypoglycemic events lacked typical sympathetic excitatory manifestations, presenting as asymptomatic hypoglycemia.

Continuous glucose monitoring was indicated after admission assessment, yet the patient declined placement due to financial concerns and fear of the monitoring device. A combined monitoring protocol of frequent fingertip blood glucose testing and venous blood glucose detection was therefore implemented. After multidisciplinary team discussion, individualized blood glucose management and nutritional support plans were formulated: adjust the infusion rate of glucose solution, raise the personalized blood glucose warning threshold to 5.0 mmol/L, and establish a tiered monitoring and closed-loop response mechanism. Sequential short-peptide enteral nutrition was initiated once hemorrhage was controlled, with synchronous monitoring of blood lactic acid and electrolyte levels.

On the 5th day of admission, melena ceased and fecal occult blood test turned negative, with hemoglobin stabilized at 95 g/L. Enteral nutrition was started on day 7, followed by transition to oral feeding on day 12. The patient was discharged on day 15 with stable clinical conditions. Discharge laboratory indicators included hemoglobin 108 g/L, blood lactic acid 2.6 mmol/L, and blood glucose fluctuating between 5.2 mmol/L and 8.6 mmol/L.

Three months of post-discharge follow-up showed no recurrent severe hypoglycemia. The patient's Barthel Index improved from 35 points (severe functional dependence) at admission to 85 points (mild functional dependence), with partial recovery of daily living abilities.

3. Discussion

This case report details a patient with coexisting mitochondrial encephalomyopathy and upper gastrointestinal bleeding who developed recurrent refractory hypoglycemia during fasting. Combined continuous intravenous fluid infusion and sequential enteral nutrition successfully maintained stable blood glucose without severe adverse events, enabling smooth hospital discharge. The nursing regimen demonstrated clear pertinence and clinical effectiveness, and the clinical challenges encountered in this case are not isolated clinical phenomena.

Existing research indicates that hypoglycemia occurs far more frequently in patients with mitochondrial disorders than in non-diabetic general populations (18.97% versus 6%), with recurrent episodes requiring early and sustained blood glucose monitoring in certain cases [8]. Furthermore, mitochondrial fatty acid oxidation defects, a subtype of mitochondrial energy metabolism dysfunction, may trigger hypoglycemia and encephalopathy under catabolic stress [9]. This further confirms that patients with mitochondrial disorders carry high hypoglycemia risks. Accordingly, targeted intervention combining glucose infusion and early enteral nutrition during fasting holds great clinical significance for this patient population.

The uniqueness and innovation of the nursing interventions in this case are reflected in rigorous clinical assessment and individualized targeted intervention. In terms of assessment, nurses promptly identified recurrent hypoglycemia in a non-diabetic fasting patient and attributed its etiology to the superimposed effects of inherent energy metabolism defects from ME and fasting-induced physiological stress. Studies have proven that patients with mitochondrial disorders commonly suffer gastrointestinal motility disorders and malnutrition, accompanied by significantly lower muscle mass and protein intake compared with healthy individuals [10]. This suggests their

limited energy reserves, which easily lead to severe metabolic decompensation under fasting stress.

In terms of intervention, this case abandoned the conventional strategy of simply increasing intravenous glucose dosage, instead establishing dual energy supply pathways combining short-peptide enteral nutrition and intravenous fluid therapy. This integrated intervention takes into account both gastrointestinal function and insulin regulatory mechanisms unique to ME patients. Published literature has verified that enteral tube feeding can effectively relieve gastrointestinal symptoms and improve nutritional status in children with mitochondrial disorders [11]. Case reports of mitochondrial neurogastrointestinal encephalomyopathy (MNGIE) also confirm that combined enteral and parenteral nutrition programs are essential to sustain patient survival [12]. In addition, extreme insulin resistance has been observed in some MNGIE patients [13], which further explains why single intravenous glucose supplementation may induce metabolic disorders. The combined nutrition strategy adopted in this case avoids such risks. These targeted adjustments reflect in-depth understanding of ME pathophysiology: short-peptide enteral nutrition alleviates absorption difficulties caused by gastrointestinal motility dysfunction, while avoiding metabolic complications arising from exclusive intravenous glucose infusion, ultimately stabilizing blood glucose and improving overall clinical prognosis.

The therapeutic mechanism of the above interventions can be explained from a pathophysiological perspective. Current research confirms that mitochondrial dysfunction directly impairs gluconeogenesis and disrupts energy metabolism, which forms the molecular basis of hypoglycemia. For instance, deficient glucose-6-phosphatase in glycogen storage disease type Ia leads to impaired oxidative phosphorylation and decreased mitochondrial membrane potential, triggering hypoglycemia [14]. Clinical data also demonstrate markedly reduced activity of mitochondrial enzymes such as succinate dehydrogenase in lymphocytes of affected patients, which strongly correlates with the severity of metabolic acidosis, confirming a direct causal link between mitochondrial damage and energy metabolism disorders [15].

In this case, the patient's upper gastrointestinal bleeding necessitated fasting, which itself exacerbates or induces hypoglycemia. Research on ketotic glycogen storage diseases has proven that fasting directly causes hypoglycemia and ketogenesis. Animal models further reveal that fasting-induced hypoglycemia promotes lipolysis in adipose tissue and inhibits the catabolism of very-low-density lipoproteins, worsening overall energy homeostasis [16]. The dual intervention of continuous fluid infusion plus enteral nutrition exerts therapeutic effects through two primary pathways:

1) Continuous intravenous glucose infusion directly provides exogenous energy substrates, bypassing the impaired endogenous gluconeogenesis pathway;

2) Amino acids, lipids and other substrates contained in enteral nutrition support hepatic and renal gluconeogenesis and β-oxidation under low-insulin conditions, generating intermediate products for the tricarboxylic acid cycle to partially compensate insufficient energy production caused by mitochondrial oxidative phosphorylation defects, stabilizing blood glucose and internal environmental balance.

Compared with standard clinical protocols, the intervention adopted in this case presents distinct advantages. Routine guidelines recommend intravenous glucose infusion as the standard correction method for hypoglycemia during fasting. However, for ME patients whose core pathological feature is impaired energy utilization, exclusive intravenous glucose supplementation cannot restore cellular energy balance effectively. In this case, the patient experienced repeated hypoglycemia despite continuous intravenous glucose administration, revealing the limitations of conventional protocols for this specific disease population. Studies on Crohn's disease patients demonstrate that enteral nutrition markedly reduces resting energy expenditure and optimizes systemic protein metabolism [17], indicating its potential to ameliorate energy utilization disorders through

metabolic regulation. Additionally, enteral nutrition represents a more physiological nutritional support modality, with unique advantages in promoting protein synthesis and regulating systemic metabolism.

Therefore, the combined enteral and parenteral nutrition strategy explored in this case conceptually surpasses the standard single intravenous glucose supplementation approach, aligning better with the metabolic characteristics of ME patients. Operationally, it ensures sustained energy supply while protecting gastrointestinal function, offering valuable references for nursing practice of similar complex clinical cases.

The identification and intervention of non-diabetic hypoglycemia in this case deliver direct implications for clinical nursing practice. When encountering non-diabetic patients with recurrent hypoglycemia, nurses must screen for rare underlying etiologies such as mitochondrial disorders. Mitochondrial dysfunction disrupts energy metabolism with highly heterogeneous clinical manifestations. Therefore, the development of nutritional regimens for fasting or perioperative patients with mitochondrial disorders must be based on underlying pathological mechanisms, adopting individualized comprehensive interventions including intravenous glucose infusion and enteral nutrition.

Multidisciplinary collaboration plays a vital role in the holistic management of metabolic disorders. Nurses must maintain close communication with physicians and clinical dietitians to address complex disease conditions and optimize patient prognosis. Moreover, continuous blood glucose monitoring and early detection of altered consciousness are critical to preventing irreversible neurological injury from severe hypoglycemia, providing new perspectives for nursing care of patients with underlying mitochondrial disorders during fasting.

As a single-center single-case report, this study lacks parallel control groups, limiting the generalizability of its findings to other patients with mitochondrial disorders complicated by acute critical illnesses. Nevertheless, this successful clinical management experience highlights a major gap in current nursing practice: standard fasting blood glucose management protocols fail to account for the core pathological feature of impaired energy utilization in mitochondrial disorder patients. Future nursing prospective studies should systematically explore the optimal timing, infusion rate and nutritional formula ratios of combined continuous intravenous fluid and sequential enteral nutrition for mitochondrial disorder patients complicated by upper gastrointestinal bleeding, infection and other acute stress states, to verify the efficacy and safety of the intervention model proposed in this case.

Given the recurrent and high-risk nature of hypoglycemia in mitochondrial disorder patients, it is urgent to establish standardized nursing protocols covering early risk screening, individualized energy support regimens and dynamic blood glucose monitoring. This creates clear directions for nursing disciplines to take a more proactive role in managing rare metabolic disorders during acute episodes.

In summary, the nursing practice of this case can be summarized as a closed-loop management framework: risk alert → comprehensive assessment → individualized targeted intervention → dynamic monitoring.

1) Maintain high vigilance for recurrent hypoglycemia in non-diabetic fasting patients, and identify its potential correlation with energy metabolism defects from mitochondrial encephalomyopathy;

2) Conduct thorough pathophysiological assessment to attribute hypoglycemia to superimposed stress of fasting and impaired gluconeogenesis caused by mitochondrial dysfunction;

3) Abandon the conventional exclusive intravenous glucose supplementation strategy, and implement individualized combined intervention of continuous intravenous fluid infusion and sequential enteral nutrition to build multiple energy supply pathways to compensate substrate

deficiency;

4) Closely monitor dynamic blood glucose levels and consciousness status to adjust intervention strategies in a timely manner based on clinical response.

This closed-loop management successfully reversed the patient's hypoglycemic crisis and underscored the importance of translating fundamental pathological mechanisms into actionable clinical nursing measures. The core value of this case lies in expanding the role of nursing disciplines in managing rare diseases such as mitochondrial disorders and their complex metabolic complications. It demonstrates nurses' initiative and scientific thinking throughout clinical assessment and decision-making, providing a replicable practical model for nursing intervention of similar metabolic emergencies.

4. Conclusion

This case report summarizes the individualized regimen of combined continuous intravenous glucose infusion and sequential enteral nutrition applied to a patient with concurrent mitochondrial encephalomyopathy and upper gastrointestinal bleeding. The protocol successfully managed recurrent hypoglycemia and prevented severe adverse events, accumulating valuable clinical experience for acute-phase nursing care of patients with special metabolic disorders.

The key clinical takeaway of this case is that frontline nurses should avoid rigid adherence to standard protocols when facing recurrent hypoglycemia in non-diabetic patients, and conduct individualized assessment based on the patient's underlying pathological mechanisms (e.g., mitochondrial energy metabolism dysfunction). This case highlights the central role of individualized comprehensive intervention in rare disease nursing. It suggests that early multidisciplinary evaluation and combined nutritional support should be initiated for similar patients to improve the scientificity of nursing clinical decision-making.

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