

# Nurse-Led Case Management for Wernicke's Encephalopathy in Acute Myelomonocytic Leukemia Post Allogeneic Stem Cell Transplantation

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**Abstract:** Wernicke's encephalopathy (WE), a neurological emergency caused by thiamine deficiency, represents a rare but life-threatening complication following allogeneic hematopoietic stem cell transplantation (allo-HSCT). This study details the nurse-led management of a 50-year-old man with acute myelomonocytic leukemia with eosinophilia (AML-M4Eo) who developed WE post-transplant. A structured nursing protocol was implemented, comprising comprehensive neurological monitoring (including consciousness and cranial nerve assessments), high-dose intravenous thiamine supplementation, individualized combined enteral and parenteral nutrition with gradual transition to oral intake, infection and bleeding prophylaxis, and psychological support using validated screening tools. After 27 days of integrated care, the patient achieved complete neurological recovery (Glasgow Coma Scale improved from 10 to 15), normalized thiamine levels (22.9 ng/mL), significant nutritional improvement, and fusion gene clearance. At the three-month follow-up, he maintained relapse-free status with substantially enhanced quality of life. This case emphasizes the vital role of systematic, nurse-driven interventions, incorporating early detection, targeted nutrient repletion, stepped nutritional rehabilitation, and psychosocial support, in optimizing WE outcomes post-allo-HSCT, supporting the integration of such multidimensional care into standard transplant protocols.

**Keywords:** Nurse-led case management; Wernicke's encephalopathy; Allogeneic stem cell transplantation; Thiamine supplementation; Nutritional support; Quality of life

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## 1. Introduction

Allogeneic hematopoietic stem cell transplantation (allo-HSCT) remains the definitive curative treatment for acute myelomonocytic leukemia with eosinophilia (AML-M4Eo), yet it is frequently complicated by metabolic and

neurological sequelae, including Wernicke's encephalopathy (WE)<sup>[1,2]</sup>. WE, a life-threatening neuropsychiatric disorder caused by severe thiamine deficiency, manifests classically as a triad of ophthalmoplegia, ataxia, and confusion, with an incidence of 0.4–2.8% in high-risk populations<sup>[3]</sup>. In the allo-HSCT setting, the risk of WE is amplified by conditioning-related catabolism, prolonged anorexia, gastrointestinal graft-versus-host disease (GVHD)-induced malabsorption, and delayed nutritional support. Despite its clinical urgency, WE is often underrecognized in transplant recipients due to atypical presentations that mimic infections, drug neurotoxicity, or GVHD-related complications, leading to delayed diagnosis and irreversible neurological damage. Current management lacks standardized nursing protocols for early detection and intervention, underscoring a critical gap in post-transplant care. Here, this study presents a case of AML-M4Eo in which a patient developed severe protein-energy malnutrition and WE following allo-HSCT. Through a structured, nurse-led approach, incorporating rigorous neurological monitoring, prompt thiamine repletion, phased nutritional rehabilitation, and psychological support, the patient achieved full neurological recovery and metabolic stabilization within 27 days. This report highlights the pivotal role of systematic nursing strategies in mitigating WE-related morbidity in high-risk transplant populations and proposes a framework for proactive surveillance and intervention.

## **2. Case presentation**

A 50-year-old male with acute myelomonocytic leukemia (AML-M4Eo, intermediate-risk; CBF $\beta$ -MYH11 positive, with KIT and NF1 mutations) was admitted on March 25, 2025, due to progressive anorexia and nutritional deterioration over two months, worsening in the past week, having been diagnosed 10 months prior with remission after induction chemotherapy but persistent fusion gene positivity, followed by matched sibling allogeneic hematopoietic stem cell transplantation (HLA 12/12 matched, donor O positive to recipient A positive) with successful engraftment, and two months before admission, he experienced abdominal pain, vomiting, and diarrhea after self-administering herbal medicine, which resolved with symptomatic treatment; on admission, he was conscious with severe anemia (hemoglobin 53 g/L), but on March 27, he developed apathy, reduced verbal output, limb weakness, and delirium with a Glasgow Coma Scale (GCS) score of 10, leading to laboratory confirmation of profound thiamine deficiency (0.012 ng/mL) and brain MRI with DWI showing restricted diffusion in bilateral thalamus and brainstem, resulting in a diagnosis of Wernicke's encephalopathy after excluding intracranial infection and transplant-associated thrombotic microangiopathy (TA-TMA); treatment involved high-dose thiamine, methylcobalamin, other B vitamins, intensive nutritional support, electrolyte correction, blood transfusions, and immunosuppressant adjustment, with nursing interventions including hourly GCS monitoring, fall prevention for ataxia and weakness, phased enteral nutrition with refeeding syndrome monitoring, bleeding risk control during hematochezia episodes (noted on April 8 with intermittent blood-stained stools), psychological support using PHQ-9 (score: 14), and medication safety protocols, leading to consciousness clearance by April 2, significant thiamine level improvement by April 3, transition to nasogastric enteral nutrition on April 12 after bleeding control, and by April 15, CBF $\beta$ -MYH11 fusion gene was negative with complete donor chimerism (100%), culminating in discharge on April 21 and no recurrence during 3-month follow-up.

## **3. Nursing interventions**

### **3.1. Systematic multidimensional monitoring**

Neurological function was monitored hourly using the Glasgow Coma Scale (GCS) to dynamically track

symptoms of Wernicke's encephalopathy, including ophthalmoplegia, ataxia, confusion, and atypical manifestations such as speech impairment. Pupillary size, light reflex, and signs of increased intracranial pressure (e.g., headache, vomiting) were documented regularly. Changes in muscle strength and language function were recorded to objectively evaluate therapeutic progress. A structured "bleeding-infection" dual-risk checklist was implemented, with handover items focusing on stool color, texture, and volume for early detection of gastrointestinal bleeding. Standardized oral care (three times daily), perineal care (twice daily), and back patting were performed to promote sputum excretion and assess infection signs in critical areas. Strict aseptic techniques were maintained during central venous catheter procedures to minimize iatrogenic infection risks. Vital signs were measured every four hours with continuous blood oxygen saturation tracking. Daily monitoring of electrolytes, blood glucose, and liver and kidney functions was conducted, and parenteral nutrition formulas and infusion rates were dynamically adjusted to prevent refeeding syndrome and metabolic disorders.

### **3.2. Precision medication management centered on thiamine supplementation**

A two-stage thiamine supplementation protocol was implemented based on EFNS guidelines <sup>[3]</sup>. During the rescue phase (March 27–29), 200 mg of thiamine dissolved in 100 mL normal saline was intravenously infused every 8 hours via pump control over 30 minutes. In the consolidation phase (March 30–April 21), 100 mg of thiamine was administered intramuscularly twice daily. Serum thiamine levels were monitored weekly, and doses were adjusted dynamically based on improvements in neuropsychiatric symptoms (e.g., apathy, delirium, speech function), transitioning from a standardized to an individualized regimen. A strict "thiamine before glucose" sequence was enforced, requiring double verification by two nurses to prevent exacerbation of thiamine deficiency due to glucose metabolism. Safety measures included readily available epinephrine for allergic reactions during intravenous thiamine administration <sup>[4]</sup>. Blood glucose, serum triglycerides, and liver and kidney functions were monitored during parenteral nutrition. For immunosuppressants (cyclosporine, mycophenolate mofetil), double verification ensured accuracy in administration time and dosage, with therapeutic drug monitoring to guide regimen adjustments.

### **3.3. Phased nutritional support**

A stepwise nutritional support plan was developed collaboratively by nurses and dietitians <sup>[5]</sup>. In the initial phase (March 27–April 7), parenteral nutrition was combined with controlled oral intake (50 mL rice broth) to prevent refeeding syndrome. During the bleeding phase (April 8–11), oral intake was halted, and pure parenteral nutrition was prioritized alongside acid-suppression therapy. In the transition phase (April 12–17), enteral nutrition was initiated via nasogastric tube at 50 mL/h and gradually increased to 100 mL/h after the patient achieved Grade II on the Kubota Water Swallowing Test. In the recovery phase (April 18–discharge), oral nutritional supplements were introduced alongside enteral nutrition, with a final transition to full oral intake of thiamine-rich foods (e.g., animal liver, whole grains) <sup>[6]</sup>. Serum albumin, prealbumin, and 24-hour fluid balance were monitored to assess tolerance and adjust nutritional formulas.

### **3.4. Multidisciplinary collaborative comprehensive intervention**

Psychological interventions were guided by Patient Health Questionnaire-9 (PHQ-9) scores, including structured relaxation training (e.g., progressive muscle relaxation, mindfulness meditation) conducted twice daily for 15 minutes to reduce eating anxiety and improve sleep quality <sup>[7,8]</sup>. Family members were engaged in caregiving

through health education manuals to enhance support. Safety measures included 24-hour accompaniment, elevated bed rails, and increased ward rounds to prevent falls and bed egress. Repositioning was assisted every two hours using a pressure-relieving air mattress to avoid pressure injuries. Daily assessments of muscle strength and activity tolerance were conducted, with guidance for progressive active and passive exercises to aid functional recovery and prevent deep vein thrombosis. A nurse-led multidisciplinary team (MDT) with a 30-minute rapid response protocol facilitated consultations (e.g., for gastrointestinal bleeding) to adjust treatment plans promptly. For blood transfusions in this ABO-incompatible transplant recipient, donor-compatible blood products (e.g., O<sup>+</sup> leukocyte-depleted red blood cells, single-donor platelets) were used <sup>[9]</sup>. A comprehensive transfusion safety process was implemented, including pre-transfusion double verification, intra-transfusion leukocyte filtration with vital sign monitoring, and post-transfusion lactate dehydrogenase (LDH) tracking to detect occult hemolysis. The 36-Item Short Form Health Survey (SF-36) was incorporated to evaluate intervention effectiveness and quality of life.

### 3.5. Standardized continuity of care

Before discharge, a personalized nutrition and medication plan was developed, emphasizing thiamine-rich foods, dosage management, and adverse reaction monitoring. A multidisciplinary follow-up team dynamically adjusted rehabilitation based on patient feedback and physiological indicators. Remote monitoring via mobile apps tracked nutritional intake and psychological status. Illustrated health education manuals and family lectures were provided to improve compliance. Collaboration with community health centers ensured the delivery of home-community care services, maintaining post-discharge continuity and effectiveness <sup>[10]</sup>.

## 4. Nursing outcomes

After 27 days of targeted, evidence-based nursing interventions, the patient showed comprehensive and sustained clinical improvements. Quantitative indicators, safety outcomes, and subjective feedback collectively validated the care plan's efficacy. Neurologically, the Glasgow Coma Scale (GCS) score improved from 10 points, marked by apathy, speech inability, and delirium, to 15 points, indicating full recovery of consciousness, clear verbal communication, and normal muscle strength and coordination. The core pathogenic indicator, serum thiamine, increased from 0.012 ng/mL (severe deficiency) to 22.894 ng/mL, returning to the normal range. Nutritionally, the severe malnutrition state was reversed: serum albumin rose from 21.82 g/L to 38 g/L, hemoglobin improved from 53 g/L (severe anemia) to 78 g/L, and prealbumin increased from 105 mg/L to 250 mg/L, with a stable 24-hour fluid intake and output balance. Psychologically, the Patient Health Questionnaire-9 (PHQ-9) score decreased from 14 points (moderate depression) to 5 points, and the 36-Item Short Form Health Survey (SF-36) score improved by 52 points, indicating a significant enhancement in overall quality of life. In terms of safety and complication control, gastrointestinal bleeding was effectively controlled within 72 hours without recurrence; no iatrogenic complications such as infection (body temperature maintained at 36.2–37.2 °C, white blood cell count stable at  $4.0\text{--}6.5 \times 10^9/\text{L}$ ), pressure injury, falls, or deep vein thrombosis occurred during hospitalization. For the ABO-incompatible transplant blood transfusion, 6 units of O<sup>+</sup> leukocyte-depleted suspended red blood cells and 2 units of single-donor platelets were administered safely, with no adverse reactions such as hemolysis, and lactate dehydrogenase (LDH) remained within the normal range (120–250 U/L). Subjectively, the patient reported, “Anxiety about neurological symptoms and bleeding has completely disappeared; I can eat and sleep normally and move freely without restrictions”. Family members noted, “Nurses’ detailed guidance on daily care and nutrition

helped us master home care skills, and remote follow-up via mobile APP gave us peace of mind”. A 3-month post-discharge follow-up confirmed stable neurological function, persistently normal nutritional indicators, negative CBF $\beta$ -MYH11 fusion gene, and 100% donor chimerism, with no disease recurrence.

## 5. Discussion

This case underscores the critical role of systematic nursing interventions in managing complex metabolic encephalopathies following allogeneic hematopoietic stem cell transplantation. The rapid neurological deterioration observed in this patient, characterized by plummeting thiamine levels and declining consciousness, highlights the time-sensitive nature of Wernicke’s encephalopathy management in post-transplant settings. Our findings demonstrate that structured nursing surveillance protocols, particularly hourly neurological assessments coupled with nutritional monitoring, can serve as early warning systems for metabolic crises that might otherwise be overlooked in complex hematological patients. The success of this intervention challenges conventional paradigms that often prioritize hematological parameters over neurological and metabolic monitoring in post-transplant care <sup>[11]</sup>. By implementing a “thiamine before glucose” protocol alongside phased nutritional support, this study addressed the fundamental pathophysiology of Wernicke’s encephalopathy while preventing iatrogenic exacerbation through glucose administration. This approach represents a significant advancement in nursing management of transplant complications, emphasizing the need for metabolic awareness in patients with nutritional deficiencies. The integration of psychological assessment using validated tools like PHQ-9 further illustrates the holistic approach required for these complex cases. The moderate depression score identified upon admission underscores the interconnectedness of metabolic, neurological, and psychological dimensions in post-transplant recovery, an aspect often underemphasized in conventional hematological care. While our results are promising, they should be interpreted within the context of single-case limitations. The absence of controlled comparisons prevents definitive causal attributions, and the unique characteristics of this case (including the specific transplant type and absence of active GVHD) may limit generalizability. Nevertheless, the temporal association between nursing interventions and clinical improvement, along with the biological plausibility of our approach, suggests genuine therapeutic efficacy.

## 6. Conclusion

This case illustrates that nurse-led, protocol-driven care models can significantly impact outcomes for patients developing Wernicke’s encephalopathy following hematopoietic stem cell transplantation. The implementation of structured neurological surveillance, metabolic monitoring, and nutritional support protocols represents a replicable framework for managing this serious complication. Our experience suggests that early recognition of nonspecific neurological changes, coupled with immediate thiamine repletion and careful nutritional management, can alter the clinical course of this potentially devastating condition. These findings advocate for greater integration of neurological and metabolic monitoring standards in post-transplant care protocols, emphasizing nursing’s pivotal role in detecting and managing these complex interdisciplinary challenges. Future research should focus on validating risk assessment tools and standardized intervention protocols across multiple centers to establish evidence-based guidelines for preventing and managing metabolic encephalopathies in high-risk transplant populations.

## Disclosure statement

The authors declare no conflict of interest.

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