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CASE REPORT

Nutritional management of a patient with acute myeloid leukaemia complicated by gastrointestinal symptoms and typhlitis

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Leukaemia is the 11th most prevalent cancer worldwide, with acute myeloid leukaemia (AML) representing an acute and highly aggressive subtype that requires urgent medical intervention. Although national data on incidence and mortality in South Africa remain limited, an epidemiological study conducted between 2015 and 2019 identified AML as the most common form of leukaemia in the country. This case study describes a 57-year-old male newly diagnosed with AML, presenting with multiple comorbidities including poorly controlled type II diabetes and class II obesity. The report underscores the complexities of nutritional management in patients with AML, as well as the unpredictable and severe complications arising from high-dose chemotherapy. These challenges significantly influence nutrition delivery and the ability to meet individual nutritional requirements.

Keywords: gastrointestinal complications in haematology, haematology nutrition, nutritional management in typhlitis, typhlitis in acute myeloid leukaemia

Introduction

A 57-year-old male patient presented to the Emergency Department with progressive fatigue over a period of six months. He reported easy bruising for a week and complained of a swollen foot. Comorbidities included:

- Poorly controlled type II diabetes
- Polyarthritis
- Peripheral neuropathy
- Mood disorder
- Hypertension
- Hypercholesterolemia
- Allergic rhinitis
- Multiple surgeries (appendectomy, hernia repair, L5–S1 fusion)
- Obesity class 2 (height: 1.77 m, weight: 118 kg, BMI 37.7 kg/m^2)

Clinical assessment

A peripheral blood sample was taken. The patient had a high white cell count (WCC) and a low platelet count (PC) (Table 1). His blood glucose level was 16 mmol/l (millimoles per litre). A high WCC with low PC can be an indication of leukaemia. Myeloid leukaemia is a cancer of young white blood cells. Immature white cells grow fast and uncontrollably, building up in the bone marrow. Acute myeloid leukaemia (AML) progresses aggressively and quickly, needs immediate attention and cannot be left untreated.[1–4]

On admission, further investigations are needed to ensure individualised, safe, and standardised treatment. The patient was started on Hydrea (hydroxyurea), a chemotherapy drug that interferes with cancer cell growth, therefore aiding in the reduction of white blood cells. The patient was also placed on an insulin sliding scale. Table 2 lists some of the ward protocols implemented for this patient.[1,2,4,7]

Days 2–6 of admission

Dietetic screening is part of the ward protocol and all patients are seen within 1–2 days of admission. According to the malnutrition risk screening tools Malnutrition Universal Screening Tool (MUST) and the National Risk Screening Tool (NRS-2002) the patient was at risk of malnutrition, but did not have any phenotypic criteria according to the Global Leadership Initiative on Malnutrition (GLIM) and therefore was not malnourished at the time of diagnosis. Acute myeloid leukaemia patients are at risk of malnutrition due to various factors (Figure 1) and therefore ongoing monitoring of nutritional risk should be undertaken. Although there is consensus on the fact that haematology patients are hypercatabolic, there are no specific nutritional recommendations for this population. The patient was eating very well and was meeting requirements as calculated. The patient's nutritional requirements were calculated according to the simplified bodyweight formula, oncology, and diabetes recommendations by the European Society for Parenteral and Enteral Nutrition (ESPEN).[11,15,32]

- Protein equivalents 1.5–1.8 g/kg/day.
- Total energy 20–25 kcal/kg/day IBW.
- Glutamine 30 g/day orally.
- 6 g oral butyric acid.

Glutamine and butyric acid are prescribed as described in Table 2.

By day four of admission the patient had gained 3 kg in weight due to fluid retention. He had a raised urea level (Table 2) and his Hemoglucotest (HGT) remained between 10 and 15 mmol/l, both of which are common side effects of cortisone treatment. He had a persistent tachycardia. Blood results showed the WCC was on a downward trend, but the infection markers (CRP) were elevated (Table 1). Cortisone increases HGT by increasing gluconeogenesis and decreasing glucose absorption in body tissue.[13,33] Cortisone causes increased catabolism of amino acids and releases nitrogen, which is converted to urea in the liver leading to high urea blood levels.[34]

Table 1: Summary of abnormal biochemical values during days 1–13 of admission

Admission day	1	4	5	6	7	8	9	10	11	12	13
WCC: 4.00–12.00 × 10^3/ul	180	135	77.6	47.4	16.5	3.5	0.1	0.2	0.2	0.1	0.1
ANC: 2.00–7.50 × 10^3/ul		1.22	0.78	0.35	0.23	0.1	0.03	0.05	0.05	0.02	0.02
Hb: 13.8–18.80 g/dl		9	8.6	8.2	8.4	7.2	6.7	7.7	8.4	8.0	8.4
Pl: 150–450 × 10^3/ul	17	12	46	35	21	12	11	28	16	48	25
CRP: 0–10 mg/l		195	169	137	92			17	11	11	43
PCT: < 0.05 mg/ml		0.56									0.5
K: 3.5–5.3 mmol/l					5.7	5.3			5.2		
Urea: 2.8–7.2 mmol/l		9.5	9.5	7.6	8.7	9.9	10.2	10.4	9.3	8.0	7.6
GFR: > 90 ml/min/1.73		69	82	83		86					
Alb: 35–57 g/l		33		33		34					
LDH: 140–271 U/l		479		412		289					

WCC: white cell count, ANC: absolute neutrophil count, Hb: haemoglobin, Pl: platelets, CRP: c-reactive protein, PCT: procalcitonin, GFR: glomerular filtration rate, Alb: albumin, 10^3/ul: thousands per microlitre, g/dl: grams per decilitre, mg/l: milligrams per litre, mmol/l: millimoles per litre, µm/l: micromoles per litre, ml/min/1.73: millilitres per minute per 1.73 square metres, g/l: grams per litre.

A family meeting was held to discuss the way forward. This is important, to explain the hospital length of stay, multiple admissions, treatment options, risks involved, possible side effects, to manage patient and family expectations, discuss consent, and to discuss possible donors as most AML patients need a hematopoietic stem cell transplant (HSCT) once remission is achieved. A peripherally inserted central catheter (PICC) line was placed for chemotherapy administration. Hydrea was decreased and the patient was due to start induction chemotherapy, which is a complex treatment plan that requires admission to hospital and continuous monitoring (Figure 2).

At this point the patient was eating well, but only meeting 60–80% of his calculated nutritional requirements. Due to the factors mentioned in Figure 1, a high-protein, fish-oil-containing, diabetic oral nutritional supplement (ONS) was prescribed for the deficit. The chosen ONS was high monounsaturated fatty acids (MUFA) and medium chain triglycerides (MCT). It contained fish oil, a mixed fibre blend, a low glycaemic index and micronutrients. Slow-release carbohydrates improve glycaemic control.

Days 7–11 of admission

The patient still had persistent tachycardia and hyperglycaemia (HGT 8–18 mmol/L). The infection markers decreased to 11 mg/l. He developed tumour lysis syndrome (TLS) with hyperkalaemia and a TLS protocol was initiated. His metformin was changed to Jalra as metformin can increase the risk of lactic acidosis in TLS. His urea levels remained elevated. The patient was now cytopenic (Table 1) with a significant decrease in WCC, PC, absolute neutrophil count (ANC), and haemoglobin (Hb) level. He required daily multiple blood and platelet transfusions. He had more than 32 items on his prescription chart, which explains the risk of fluid retention and aversion to any additional oral prescriptions.

Days 12–14 of admission

On the evening of day 11 the patient became pyrexial, hypertensive, and his HGT increased to 20 mmol/l. On day 12 his IV fluids were decreased, Lasix was started, cortisone was stopped and cytarabine finished (he was still on venetoclax as per Figure 2). He developed diarrhoea. The dietitian changed the menu to a gastro diet and the ONS to a hydrolysed, prebiotic containing high-MCT product. The chosen ONS is easily digested and absorbed and better tolerated in patients with diarrhoea due to the peptide-based, 100% whey protein composition. The prebiotics support the compromised gut microbiome. This product also has a lower osmolarity than many energy-dense ONSs, preventing osmotic diarrhoea in a compromised GIT. Additional micronutrients provide immune-modulating effects, and also provide additional calories and protein to meet the needs of a critically ill patient. Glutamine and butyric acid was continued. In the afternoon of day 14 the patient's condition deteriorated with hypotension, pyrexia (40°C), hyperglycaemia, tachycardia, abdominal distention, and severe diarrhoea. Antibiotic cover was increased, the PICC line was removed, cortisone was restarted and he was transferred to the intensive care unit (ICU). An abdominal sonar was done, but no abnormalities were found.

ICU days 2–6

The patient had wide-ranging changes on a daily basis as summarised in Table 3.

Typhlitis

Typhlitis (neutropenic colitis) is a complication that mostly affects patients receiving high-dose chemotherapy. Mucosal barrier injury together with bone marrow suppression caused by the treatment increases the risk of typhlitis.[35] Certain drugs that increase the risk of typhlitis include daunorubicin, cytarabine, and broad-spectrum antibiotics (cefepime, meropenem), all of which this patient received. High-dose chemotherapy causes mucosal barrier injury and necrosis of various layers of the intestinal mucosa. Antibiotics disrupt the normal balance of the gut microbiome. Glutamine can be given to support mucosal barrier injury, but oral glutamine does not reach the colon. Probiotics can be prescribed to support dysbiosis, but they are contraindicated in patients with neutropenia.[21,36]

Symptoms include abdominal pain in the lower right quadrant (terminal ileum/caecum), abdominal distention or discomfort and watery or bloody diarrhoea. Typhlitis can be complicated by obstruction, ileus, and, in severe cases, perforation. Diagnosis is made with an abdominal computed tomography (CT) scan. Gut rest is the first step in treating typhlitis, with a nasogastric tube (NGT) for drainage. Patients receive nutritional support with total parenteral nutrition (TPN). Medical management includes targeted antibiotics (blood cultures and stool samples are taken to determine the infective organism) and fluid management.[35,37]

Table 2: Bone Marrow Unit (BMU) admission protocols with specific reference to the patient

Prescription	Rationale	Additional information	Ref
Cardiac investigations ECG (echocardiogram)	By determining the heart function the doctors can prevent cardiotoxicity or adjust dosages to prevent exacerbation of existing conditions	These indicators are important to calculate a safe dosage for the patient	2,5
Bone marrow aspiration test (BMAT)	To confirm the diagnosis	This is necessary to identify the type of AML, as there are > 7 subtypes. Genetic or chromosomal abnormalities also need to be investigated to ensure targeted treatment	4,6,7
Hydrea	Decreases white cell count by inhibiting cell proliferation	Short-term treatment for high WCC. Hydrea targets fast-dividing cells, it increases the risk of oral mucositis	4,8
Empiric antibiotics	In AML the white blood cells, although present in large volumes, are immature and unable to fight infection	Prophylactic antibiotics decrease the risk of bacterial and invasive fungal infections	3,9
Multivitamin	A standard multivitamin is provided to ensure patients meet (at least) RDA for micronutrients	HD chemotherapy increases cell proliferation and micronutrients are metabolized faster than in healthy individuals	10,11
Cortisone	Cortisone is prescribed to manage nausea Cortisone also decreases WCC, inflammation and improves treatment efficacy	Cortisone increases the risk of hyperglycaemia and gastritis, and leads to raised urea levels	5,12,13
PPI	Cortisone increases the risk of gastritis. HD chemotherapy increases the risk of heartburn, MBI, and GIT bleeds	PPI affects absorption of medication. H2 receptor blockers ensure improved absorption of medication and electrolytes, but are less effective in preventing symptoms	14,15
Antiemetics	Prophylactic antiemetics are prescribed to prevent nausea, which is a common side effect of HD chemotherapy	In-hospital treatment means patients have IV access and a variety of drugs can be given to target all types of nausea (acute, delayed, breakthrough, refractory)	15,16
Fluids, puricos, allopurinol, sodium bicarbonate	HD chemotherapy leads to increased haemolysis (destruction of blood cells). Leukostasis (high WCC) leads to obstruction of blood flow. This can lead to AKI, increased uric acid and lactic acidosis	Alkalizing agents decrease the risk of lactic acidosis and renal injury	17,18
The medical team considered Leukapheresis	The removal of abnormal white blood cells through apheresis	Uncontrolled multiplication of white cells can lead to leukostasis, thrombin formation, or microangiopathy	18,19
Mouthwashes: anti-bacterial and in-house mixture (sodium + anaesthetic mix)	Chemotherapy targets fast-dividing cells; this includes the mucosa of the mouth. Xerostomia increases the risk of infection	Saliva maintains oral health by cleansing, neutralising, and protecting against microbial infections	6,20–22
Neutropenic hospital diet	Severe neutropenia with MBI increases the risk of microbial translocation. Educating patients on food safety, personal hygiene, safe food handling, and safe drinking water is important	The Neutropenic Diet has been controversial and is specific to each unit. In the South African setting, with multiple food-borne diseases and deaths, socioeconomic disparity, and lenient food labelling and production laws this is an area that cannot be ignored. Clean and safe water, toilet facilities, and maintaining personal hygiene are not accessible to all patients	23–25
Daily weight	High volumes of IV infusions, TLS and AKI can lead to fluid retention	Daily weighing can help identify fluid retention before complications arise and may even improve patient outcomes	26
Glutamine	In MBI supporting the enterocytes is important. It reduces mucositis severity, and bacterial translocation, increases citrulline production and is fuel for enterocytes	Glutamine is conditionally essential and in patients where MBI is expected can be beneficial in reducing severity of symptoms. Oral Glutamine is, however, used up before reaching the colon	27
Butyric acid	Dysbiosis of the microbiome, MBI, and Neutropenia increase the risk of microbial translocation and sepsis. SCFAs improve epithelial development, regulate crypt depth, have a trophic effect on colonocytes, and promote intestinal repair and colonic T-cell expansion	Prebiotics are metabolized into SCFAs by probiotics Probiotics are contraindicated in neutropenic patients, thus this process cannot be supported by supplementing pre- or probiotics	28–31

AML: acute myeloid leukaemia, WCC: white cell count, RDA: Recommended Dietary Allowance, HD: high dose, MBI: mucosal barrier injury, GIT: gastrointestinal tract, PPI: proton pump inhibitors, H_2: hystamin-2, IV: intravenous, AKI: acute kidney injury, TLS: tumour lysis syndrome, SCFA: short-chain fatty acids.

ICU Day 6 nutritional prescription

According to Lopez-Delgado et al.,[11] haematological malignancy treatments are associated with amplified oxidative stress, greater systemic inflammation, and a high risk of a decline in nutritional status. ICU stay of more than 48 hours increases the risk of malnutrition in this patient population as fluid and electrolyte imbalances and a calorie deficit increase muscle catabolism and sarcopenia. By using predictive equations for ICU patients, the current patient's requirements were recalculated:[10,11,14,31,37,38]

- Macronutrients:
  - Total energy: 25–30 kcal/kg/day.
  - Protein equivalents: 1.3–1.8 g/kg/day.
    - Glutamine: 0.35 g/kg/day.
  - Carbohydrates: 60% of non-protein energy (NPE).
  - Fat: 40% of NPE.
    - Omega 3: 1.8–2 g/day.

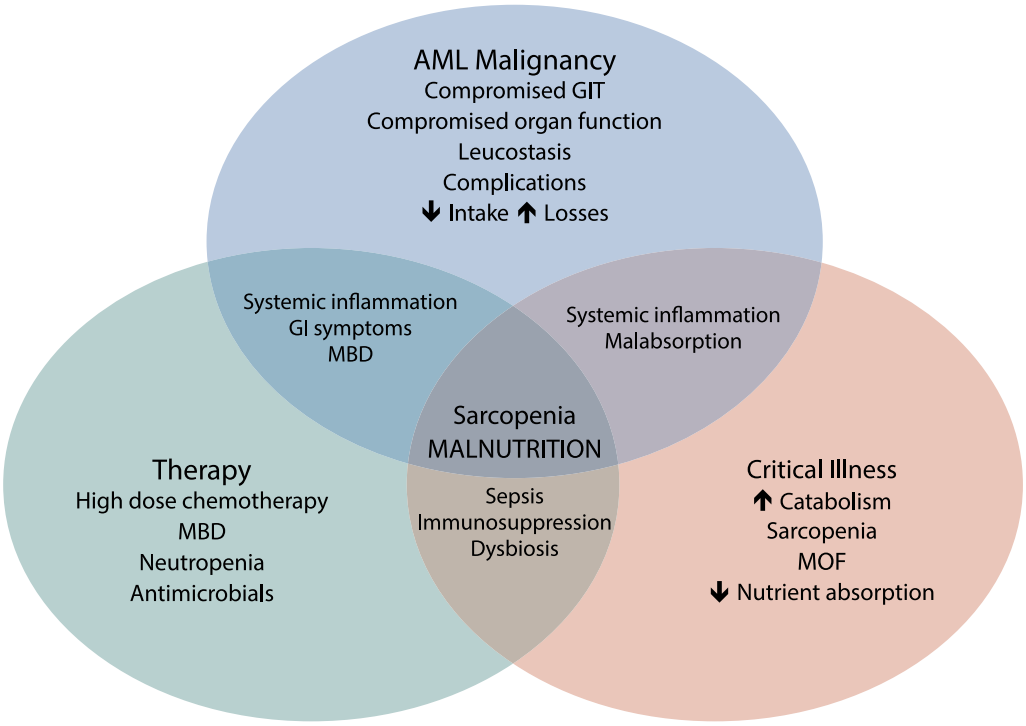

Figure 1: Factors that increase the risk of malnutrition in AML patients. Image adapted from Lopez-Delgado et al.[11] AML: acute myeloid leukaemia, GI: gastrointestinal, MBD: mucosal barrier damage, MOF: multi-organ failure, ↑ increased, ↓ decreased.

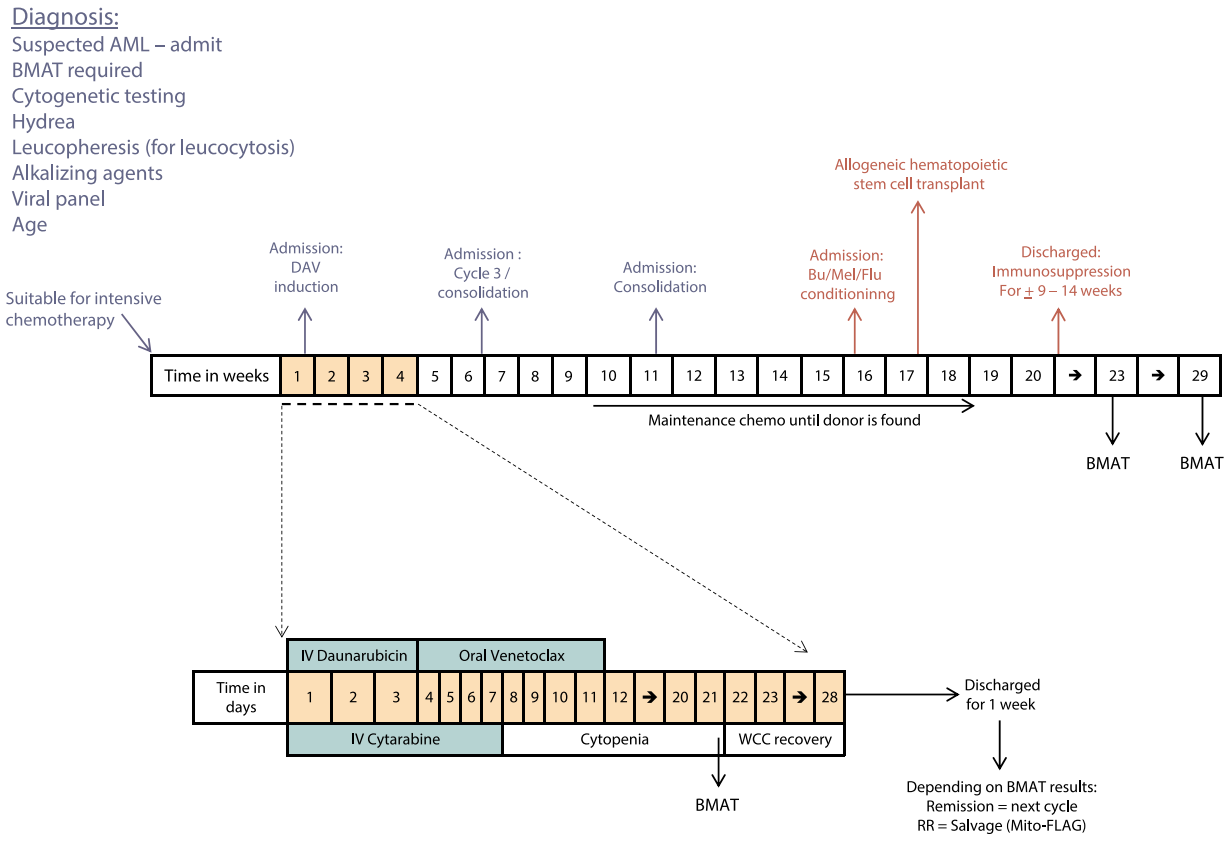

Figure 2: Ideal AML treatment plan, if a patient is deemed suitable for intensive treatment. This treatment regimen can change at any time, depending on the patient's tolerance, complications during treatment, or delay in finding a suitable donor. AML: acute myeloid leukaemia, BMAT: bone marrow biopsy test, DAV: daunorubicin–cytarabine–venetoclax, Bu/Mel/Flu: busulfan–melphalan–fludarabine, IV: intravenous, WCC: white cell count, RR: relapsed or refractory, Mito-FLAG: mitoxantrone–fludarabine–cytarabine–growth factors.

Table 3: Summary of clinical presentation, abnormal biochemical values, events, and interventions during days 2–6 in ICU

ICU	Day 2	Day 3	Day 4	Day 5	Day 6
Clinical	Pyrexial Tachycardia Hypertensive Abdominal distention HGT 7–16 mmol/l	Apyrexia Tachycardia Fluid balance +1L HGT 7–16 mmol/l	Apyrexia Tachycardia Anorexia Diarrhoea settled HGT 8–16 mmol/l	Apyrexia Tachycardia Abdominal pain HGT 5–10 mmol/l	Pyrexia Tachycardia Severe abdominal pain Unable to eat
Biochemistry Normal values (Oncolab)					
WCC: 4.00–12.00 × 10^3/ul ANC: 2.00–7.50 × 10^3/ul Hb: 13.8–18.80 g/dl Pl: 150–450 × 10^3/ul	WCC 0.1 ANC 0.01 Hb 7.3 Pl 7	WCC 0.1 ANC 0.01 Hb 7.5 Pl 19	WCC 0.1 ANC 0.01 Hb 9.2 Pl 16	WCC 0.1 ANC 0.12 Hb 9 Pl 3	WCC 0.1 ANC 0.01 Hb 9 Pl 14
CRP: 0–10 mg/l PCT: <0.05 mg/ml	CRP 470 PCT 83	CRP 453 PCT 40	CRP 414 PCT 16	CRP 367 PCT 10	CRP 413 PCT 9.2
Na^+: 136–145 mmol/l Cl^-: 98–107 mmol/l Urea: 2.8-7.2 mmol/l Creat: 64.00–104.00 µmol/l GFR: > 90 ml/min/1.73	Na 133 Cl 97 Urea 10.7 Creatinine 157 GFR 42	Urea 9.3 GFR 78	Urea 8.6	Urea 11.2 GFR 89	Urea 11.1
Alb: 35–57 g/l	Alb 29	Alb 27	Alb 27	Alb 26	Alb 25
Medical interventions	Antibiotics New CVC Inotropic support Insulin sliding scale Antidiarrheal Rx Blood + platelet transfusion	Diuretic Decrease IV fluids Fluconazole added to increase venetoclax effectiveness	Wean inotropes and cortisone Platelet transfusion	Platelet transfusion Consider transferring patient to the ward	Abdominal CT showed bowel obstruction, steatosis, enteritis, typhlitis NPO NGT inserted and drained 1 200 ml Referral to surgeon TPN IV Glutamine
Dietetic input Early ICU admission guidelines	Requirements remained the same Gastro/DM diet – patient was still eating Hydrolysed ONS od–bd SCFA 3 g bd		Gln orally 30 g/day Change ONS to high protein, diabetic products bd–tds		

ICU: intensive care unit, HGT: Hemoglucotest, WCC: white cell count, ANC: absolute neutrophil count, Hb: haemoglobin, Pl: platelets, CRP: c-reactive protein, PCT: procalcitonin, Na: sodium, Cl: chloride, Creat: creatinine, GFR: glomerular filtration rate, Alb: albumin, CVC: central venous catheter line, Rx: treatment, 10^3/u: thousands per microlitre, g/dl: grams per decilitre, mg/l: milligrams per litre, mmol/l: millimoles per litre, µm/l: micromoles per litre, ml/min/1.73: millilitres per minute per 1.73 square metres, g/l: grams per litre.

- Micronutrients:
  - 100% recommended dietary allowance (RDA) daily.

Total parenteral nutrition (TPN) was started. Due to inflammation and fluid limitations a TPN bag was chosen containing a mix of fats (olive oil, fish oil, soya, and medium chain triglycerides [MCT]) and micronutrients (water-soluble vitamins, fat-soluble vitamins, minerals, and trace elements). The bag did not contain any glutamine; this was added as an intravenous (IV) L-alanine-glutamine infusion. There was no contra-indication to glutamine and it formed part of the amino acid (AA) profile rather than being supplemented. Unfortunately we were only able to meet 77% of the patient's macronutrient requirements, but 100% RDA of micronutrients. The patient remained nil per os (NPO), with an NGT on free drainage.

Fluid overload or hypervolemia is a common complication in AML induction patients due to supportive transfusion of blood products as well as hyperhydration as part of the chemotherapy protocols. In ICU fluid therapy, given intravenously, is a common intervention. This patient had received induction chemotherapy, was critically ill and NPO. Therefore all drug administration was also IV.[39,40] On the prescription chart there were more than 20 IV drugs prescribed. These included:

- Nutrition: TPN and glutamine.
- Antibiotics and antiviral medication.
- Analgesics.
- Proton pump inhibitor.
- Vasopressors.
- Platelet and red blood cell transfusions.
- Hydrocortisone.

Drugs were administered in the strongest possible solution to decrease volume administration and some drugs were administered together, where it was safe to do so. This explains the fluid restriction and the reason why the TPN could not be administered at the required rate.

ICU days 7–13

The patient became confused, pulled out his NGT multiple times and it had to be replaced four times. The NGT drained 450–2 600 ml over the next few days. Despite receiving multiple blood and platelet transfusions, he presented with refractory anaemia and thrombocytopenia. Persistent tachycardia, hyperglycaemia, pyrexia, and neutropenia made the patient a poor surgical candidate (Table 4). The surgeon opted for a

Table 4: Summary of abnormal biochemical values during days 7–14 of ICU admission

ICU days	7	8	9	10	11	12	13	14
WCC: 4.00–12.00 × 10^3/ul	0.1	0.1	0.1	0.1	0.1	0.1	0.1	0.1
ANC: 2.00–7.50 × 10^3/ul	0.01	0.01	0.01	0.00	0.01	0.00	0.02	0.00
Hb: 13.8–18.80 g/dl	7.3	9.2	8.8	8.5	8.0	8.2	8.6	9
Pl: 150–450 × 10^3/ul	11	11	13	7	10	18	18	16
CRP: 0–10 mg/l	401	379	322	264	278	318	264	236
PCT: < 0.05 mg/ml	9.3	9	7.4		8.5	7.4	6	5.5
Na^+: 136–145 mmol/l								
Urea: 2.8–7.2 mmol/l	11.6	13.1	12.4	11.8	11	12.6	10.8	11.2
Creat: 64.00–104.00 µmol/l				56.7		60.5		
GFR: > 90 ml/min/1.73	23	23	21	19	19			
GGT: 9–64 U/l								88
Alb: 35–57 g/l	23	23	21	19	19	19	20	19

ICU: intensive care unit, WCC: white cell count, ANC: absolute neutrophil count, Hb: haemoglobin, Pl: platelets, CRP: c-reactive protein, PCT: procalcitonin, Na: sodium, Creat: creatinine, GFR: glomerular filtration rate, GGT: gamma-glutamyl transferase, Alb: albumin, 10^3/ul: thousands per microlitre, g/dl: grams per decilitre, mg/l: milligrams per litre, mmol/l: millimoles per litre, µm/l: micromoles per litre, ml/min/1.73: millilitres per minute per 1.73 square metres, U/l: units per litre, g/l: grams per litre.

conservative approach. A Gastrografin follow-through was ordered. A water-soluble contrast (Gastrografin) is swallowed or given via NGT. A series of X-rays are taken to study the movement of the contrast through the gastrointestinal tract.[40]

After three days the Gastrografin was still in progress. Prokinetics were added to increase gastric motility and attempt to accelerate the follow-through. By this time the patient had severe hypoalbuminemia (Table 4). Although albumin is not a good indicator of nutritional status, it is an indicator of a poor clinical condition and prognosis. In critical illness, intra- and extravascular albumin distribution is altered: synthesis is decreased and degradation increased. Albumin is a carrier of multiple substances in the blood. Therefore a significant drop may decrease efficacy and delivery of medications or lead to drug toxicity. Albumin has antioxidant potential, maintains microvascular integrity, and acts as a plasma buffer.[41,42]

By day 12 of ICU, the patient was on day 20 of his chemotherapy cycle and due for a BMAT. Unfortunately the results showed that he had residual disease (there were still 20% blasts/immature blood cells present). He was not in remission.

The clinical indicators did not change, but the CRP was on a decline. The Gastrografin contrast was still present in the bowel after six days. Another scan confirmed that there was no change in the bowel condition and the surgeon decided to do a laparotomy. During this time the patient's nutrition could not be changed due to all the limiting factors. He was still receiving only 77% of the calculated nutritional requirements.

ICU days 14–20

The surgeon found turbid fluid in the abdomen; a washout was done with adhesiolysis and repair of serosal tears. A pencil drain was inserted. Postoperatively, the patient's condition slowly improved. The infection markers decreased, the WCC recovered and the PC was increasing without transfusions. The TPN had to be changed to an electrolyte-free regimen due to raised sodium levels, which resolved after two days. NGT drainage decreased and by day 16 the NGT was removed. At this time the dietitian was able to start a progression diet with ONS, whilst the patient was still on TPN. The dietitian prescribed a clear-fluid ONS with clear fluids from the kitchen. The clear fluid ONS was high in calories with 4 g/100 ml whey protein, providing nutrients, whilst being easily digested, and fat and fibre free. He was able to progress to a full fluid diet the following day with fluids from the kitchen and the same diabetic, fish-oil containing, high-protein ONS was prescribed as in the first week of admission. By day 18 the TPN was stopped and a soft diet with the above-mentioned ONS was prescribed. Butyric acid was continued. The patient did not want to take the additional oral Glutamine supplement. He progressed to a full diet without any concerns, other than the hepatic function (Table 5).

ICU day 21

The events of this day were as follows: At 8:00 the patient was seen by the dietitian. He was sitting in bed, eating bacon and eggs for breakfast. He was positive and reported feeling very well. Shortly afterwards the haematologist saw the patient and planned for a ward transfer the next day. At 9:45 the physiotherapist (PT) came to see the patient. He was agitated and asked to be assisted onto the bedpan. At 10:00 the PT returned to assist the patient back into bed and found melena stools in the bedpan. Thereafter the nurse collected two litres of frank blood in the bedpan. At 11:00 the patient became hypotensive. He was hyperglycaemic, with a lactate level of 4 mmol/l. The surgeon came to see the patient. He was still awake, orientated, and communicating well. He was taken to theatre for a gastroscopy and a colonoscopy. He was too unstable for a laparotomy. The patient returned to ICU intubated and on inotropic support. No active bleed was seen and the surgeon reported that the bleed was most probably in the small intestine, but again the patient was too unstable to be taken for a scan. At 20:00 the doctor requested TPN. An interim three-chamber TPN bag was started at a low rate.

ICU days 22–25

The patient's condition deteriorated rapidly overnight (Table 5). His HGT varied between 3 and 16 mmol/l. He was anuric, acidotic (lactate 16–19 mmol/l), with fresh blood draining from his NGT. The nephrologist was consulted and continuous venous–venous haemodialysis (CVVHD) was started. The dietitian then consulted the medical team and requested that TPN be stopped, as the patient was haemodynamically unstable. The LFTs were still deranged (Table 5).

By day 25 the patient was in multi-organ failure (MOF). He was ventilated, sedated, and on CVVHD. Two inotropes, given at double strength (360 mcg/hour and 7.2 g/hour respectively), were not enough to improve hypotension. Furthermore the patient

Table 5: Summary of abnormal biochemical values during days 15 - 25 of ICU admission

ICU days	15	16	17	19	21	22	23	24	25
WCC: 4.00–12.00 × 10^3/ul	N/A	0.4	0.9	1.9	3.5	25	31	42	51
ANC: 2.00–7.50 × 10^3/ul	N/A	0.01	0.32	1.7	N/A	23	27	31	47
Hb: 13.8–18.80 g/dl	N/A	8.3	8.7	8.3	7.8	5.1	8.2	8.2	7.6
Pl: 150–450 × 10^3/ul	N/A	9	16	27	65	96	30	30	34
CRP: 0–10 mg/l	237	237	200	65	93	44	234	272	353
PCT: < 0.05 mg/ml	4.5	4.5	4.5	2	1.4	69	75	95	92
Na^+: 136–145 mmol/l		146							
Urea: 2.8–7.2 mmol/l	13.3	13	12.8	12	10	13.8	10	9	8
Creat: 64.00–104.00 µmol/l						255	185	168	163
GFR: > 90 ml/min/1.73						23	34	38	40
ALP: 34–104 U/l		108	258	446	582	258	498	748	762
GGT: 9–64 U/l	101	139	259	565	700	233	288	312	266
ALT: 7–53 U/l	56	62	115	182	144	1 530	1 914	1 409	1 001
AST: 13–39 U/l			94	102	77	5 355	4 962	1 409	771
Alb: 35–57 g/l	19	18	18	19	20	14	17	16	14
LDH: 140–271 U/l				291	293	6 661	7 853	2 205	1 441

ICU: intensive care unit, WCC: white cell count, ANC: absolute neutrophil count, Hb: haemoglobin, Pl: platelets, CRP: c-reactive protein, PCT: procalcitonin, Na: sodium, Creat: creatinine, GFR: glomerular filtration rate ALP: alkaline phosphatase, GGT: gamma-glutamyl transferase, ALT: alanine transaminase, AST: aspartate transaminase, Alb: albumin, LDH: lactate dehydrogenase, 10^3/ul: thousands per microlitre, g/dl: grams per decilitre, mg/l: milligrams per litre, mmol/l: millimoles per litre, µm/l: micromoles per litre, ml/min/1.73: millilitres per minute per 1.73 square metres, U/l: units per litre, g/l: grams per litre.

had now completely relapsed disease with a WCC of 51 × 10^3/ul (thousands per microlitre). The NGT continued to drain blood. No nutritional support was possible during this time. A family meeting was held and the decision was made to discontinue treatment. The patient passed away only a few hours later.

Managing nutrient demand, glycaemic control, nutritional losses, treatment complications, and fluid balance in an obese patient within an ICU setting led to compounding nutritional challenges in this case:

- An increase in metabolic demand caused by the inflammation of the disease and the chemotherapy, but worsened significantly by the intestinal inflammation and infection. Meeting these increased nutritional requirements, especially protein delivery, proved to be impossible.[11,13,40]
- Impaired nutrient absorption occurs due to the MBD, altered gut microbiota, and neutropenia due to the disease and treatment. With the added bowel damage and inflammation caused by typhlitis and its management, nutrient breakdown and absorption is impaired. The GIT could not be supported with probiotics and the supplementation of oral Glutamine and SCFA did not prevent treatment complications in this patient.[13]
- Increased losses caused by diarrhoea and nasogastric drainage leads to a nutrient deficit and electrolyte imbalances, which were aggravated by the cell destruction caused by the chemotherapy. Fortunately, due to the fact that high-dose chemotherapy patients have daily blood tests and IV access, the electrolyte imbalances can be monitored and managed.[13,33]
- In the early management of typhlitis the patient was NPO, without the possibility to feed enterally.
- Hyperglycaemia was another complication that was difficult to manage. The patient was diabetic and prescribed cortisone as part of his AML treatment protocol and later in the management of his critical illness. This was aggravated by the inflammatory response and despite all attempts to manage his blood glucose levels (low glucose provision via TPN and an insulin infusion), a normal glucose level was not maintained.[33]
- Early after admission the patient received daily, multiple transfusions of red blood cells and platelets. In ICU the addition of intravenous (IV) medications quickly led to a fluid overload. The medical team worked hard to restrict IV fluids. This restriction included the volume of TPN, which contributed to the inability to meet the patient's nutritional requirements.[40,41]

Conclusion

AML is an aggressive, life-threatening disease. Treatments are harsh and sometimes debilitating, with multiple risks. Many patients achieve remission and complete recovery after chemotherapy and HSCT. Nutritional monitoring and support aims to improve a patient's treatment tolerance, infection and complication risk, length of hospital stay, recovery, and quality of life.[6,11,33] This case highlights the complexities, unpredictability, and severe risks for haematology patients and the treatments they receive.

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