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CASE REPORT

Nutritional management of a patient with a small bowel neuroendocrine tumour and obstruction: a South African case study

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Small bowel neuroendocrine tumours (SBNETs) arise from enterochromaffin cells, most commonly in the terminal ileum. Functional tumours may secrete hormones, which can lead to carcinoid syndrome, whereas non-functional tumours often present later with symptoms of mechanical obstruction. Malnutrition is common in patients with gastroenteropancreatic neuroendocrine tumours (GEP-NETs) which includes SBNETs, and is worsened by tumour burden, medical therapies, hormonal activity, surgery, and postoperative catabolism.

A 55-year-old male presented with abdominal discomfort, early satiety, vomiting, and unintentional weight loss. Imaging revealed a small bowel mass with biochemistry tests indicating a neuroendocrine tumour (NET). Preoperative nutritional assessment revealed severe malnutrition, prompting the initiation of parenteral nutrition prior to abdominal surgery. Throughout the preoperative and postoperative period, targeted nutritional intervention contributed to improvements in fluid balance, maintenance of skeletal muscle, and an increased phase angle, all reflecting an overall improvement in the patient's nutritional status.

Malnutrition in patients with NETs is multifactorial, and early identification, targeted nutritional support, and structured follow-up are essential to optimise recovery and long-term outcomes in this population.

Keywords: bioelectrical impedance, body composition, bowel obstruction, dietetics, hepatobiliary, neuroendocrine tumour, nutrition support, oncology, phase angle, surgery

Introduction

Neuroendocrine neoplasms (NENs) comprise two distinct groups: neuroendocrine tumours (NETs) and neuroendocrine carcinomas (NECs).[1,2] NETs are uncommon malignancies that arise from neuroendocrine cells, which are distributed throughout the body.[1,2] These cells are most frequently found in the gastrointestinal tract, pancreas, and lungs, but NETs may also occur in the head and neck region, breast tissue, genitourinary tract, and skin.[1,2] Among gastrointestinal NETs, small bowel NETs (SBNETs) represent a significant subtype and are among the most common small intestinal cancers, with jejunoileal NENs showing the highest incidence compared with duodenal NENs and adenocarcinomas.[3]

NETs differ significantly from NECs, which are poorly differentiated, high-grade malignancies with aggressive behaviour and a worse prognosis.[1,2] While NETs often grow slowly and are well differentiated, NECs are typically resistant to treatment and require aggressive management such as systemic chemotherapy.[1,2]

The incidence and prevalence of NENs have risen steadily worldwide over the past decade.[3] Large population-based studies, such as the Surveillance, Epidemiology, and End Results (SEER) database in the United States, report a 6.4-fold increase in the age-adjusted incidence of all NENs between 1973 and 2012 (from 1.09–6.98 per 100 000 persons per year), reaching 8.19 per 100 000 by 2018.[3] Other high-income countries, including Canada, England, Norway, Australia, and parts of Europe, show similar upward trends, while incidence in Asia remains comparatively lower but rising.[3] This increase is thought to result from enhanced physician awareness, improved imaging and diagnostic techniques, and updates to histopathological classifications, such as the WHO 2019 and 2022 updates that introduced well-differentiated Grade 3 NETs and refined the classification of NECs.[3]

Epidemiological data from Africa, and South Africa in particular, remain scarce. Existing registries are largely pathology-based and do not differentiate NET subtypes, such as small bowel NETs, and, consequently, the true incidence and prevalence of all NETs in South Africa remains unclear.[4]

Small bowel NETs arise from enterochromaffin cells, most commonly located in the terminal ileum, with their clinical presentation being dependent on tumour functionality and the extent of the disease.[2] Functional tumours may secrete serotonin or other vasoactive hormones possibly leading to carcinoid syndrome, while non-functional tumours often remain clinically silent until they cause a mechanical obstruction, which may present as abdominal pain, altered bowel habits, vomiting or gastrointestinal bleeding.[2]

Carcinoid syndrome and carcinoid crisis

Carcinoid syndrome results from the overproduction of serotonin (5-HT), the principal mediator, along with other vasoactive substances including polypeptides, vasoactive amines, and prostaglandins which enter systemic circulation.[5] The syndrome is most commonly associated with midgut NETs, particularly in the presence of extensive liver metastases, as these substances bypass hepatic metabolism.[5] Common symptoms include facial flushing (seen in 90% of cases) and secretory diarrhoea (60–80%) with abdominal cramping due to increased intestinal motility and secretions.[2,5] Chronic exposure to these secreted substances can also result in carcinoid heart disease and a

niacin deficiency, leading to pellagra, which may present with dermatitis, worsened diarrhoea, and in severe cases, dementia.[5]

Serotonin is synthesised from tryptophan, an amino acid that is also the primary precursor for niacin synthesis.[5] In a normal context, approximately 99% of tryptophan is metabolised via the pathway producing nicotinic acid (niacin), with only a small fraction used for serotonin production.[5] In carcinoid syndrome, however, tumours can consume up to 60% of the body's tryptophan for serotonin synthesis, diverting it away from niacin production and precipitating a niacin deficiency if the syndrome is not adequately controlled.[5]

Diagnosis requires the presence of symptoms and is confirmed by elevated urinary 5-hydroxyindoleacetic acid (5-HIAA) levels; however, false-positive results related to serotonergic medications or the dietary intake of serotonin-rich food may occur.[5] Management of the syndrome includes the use of somatostatin analogues such as octreotide, which works by inhibiting hormone secretion through binding to somatostatin receptors that are expressed on most NETs.[5,6]

A rare but potentially life-threatening complication is carcinoid crisis, which can present with sudden haemodynamic instability, including severe hypotension or prolonged hypertension, which may be accompanied by the classical carcinoid syndrome symptoms.[6] It is believed to result from a massive release of vasoactive substances, often triggered by abdominal surgery although it can occur spontaneously.[6] Prophylactic administration of octreotide preoperatively is a key component of management to reduce the risk of this complication occurring.[6]

Diagnosis

The diagnosis of neuroendocrine tumours is done through a combination of imaging, biochemical, and histological assessments. Imaging typically includes computed tomography (CT), magnetic resonance imaging (MRI), and positron emission tomography-computed tomography (PET-CT). [2] Among these modalities, ^{68}Ga-labelled somatostatin analogue PET/CT (e.g. ^{68}Ga-DOTATOC) is considered the new gold-standard functional imaging technique for neuroendocrine tumours, providing high sensitivity and specificity for tumour localisation and staging.[2] Biochemical assessments include testing serum Chromogranin A (CgA) and 24-hour urinary 5-HIAA, which reflect tumour burden and serotonin production, respectively.[2] A definitive diagnosis however, is only obtained via biopsy with immunohistochemical analysis.[2]

Nutritional implications

Nutritional complications in patients with gastroenteropancreatic neuroendocrine tumours (GEP-NETs) are multifactorial, with 14–38% of patients reported to be at risk of malnutrition.[1] Among those receiving somatostatin analogue therapy, malnutrition has been observed in up to 75% of cases in a recent study, with sarcopenia present in approximately 70% of these.[7] The presence of malnutrition additionally demonstrated a significantly worse overall survival.[7]

In patients with SBNETs in particular, tumour-related factors such as bowel obstruction and impaired absorption can lead to both macro- and micronutrient deficiencies.[1] Hormonal disturbances also play a role, particularly in functional tumours where carcinoid syndrome is most frequently associated with SBNETs and can lead to gastrointestinal symptoms, with associated increased nutrient losses and a reduction in nutritional intake and assimilation.[1,5] Additionally, GEP-NETs have been linked with deficiencies in fat-soluble vitamins, particularly vitamin D.[1,7] Moreover, medical therapies such as somatostatin analogues, while effective for carcinoid syndrome symptom control, can induce exocrine pancreatic insufficiency in up to 20% of cases, impacting fat and fat-soluble vitamin malabsorption further.[1,6] Surgical resection is another major contributor to nutritional compromise. The extent of small bowel resection during surgery can significantly influence nutrient absorption, with larger resections increasing the risk of malabsorption and subsequent nutrient losses. The specific site of resection also plays a role, as different regions of the small intestine are responsible for the absorption of distinct nutrients. In addition, the catabolic stress response that typically occurs in the immediate postoperative period further compounds nutritional compromise, increasing protein and energy requirements while reducing overall reserves.[1,8]

Collectively, these factors highlight the high risk of malnutrition in small bowel NETs and the importance of timely nutritional assessment, monitoring, and targeted nutritional interventions throughout the course of disease and treatment.

Case presentation

A 55-year-old male with a history of type 2 diabetes mellitus and hypertension presented to the hepatopancreaticobiliary (HPB) surgical ward with a five-month history of abdominal discomfort, early satiety, post-prandial vomiting, and significant unintentional weight loss.

A CT scan performed in February 2025 revealed a small bowel mass partially obstructing his bowel, with multiple bilobar liver lesions and an elevated CgA level in his blood, which supported the diagnosis of a neuroendocrine tumour.

There was no clinical evidence of carcinoid syndrome, and his urinary 5-HIAA levels were within normal limits.

Nutritional assessment

Dietetic assessment was first conducted on May 6, 2025. At this time, he weighed 63.3 kg with a height of 1.66 m (body mass index [BMI] 22.9 kg/m^2), reporting an involuntary loss of 30 kg (~32%) from his pre-morbid weight of 93 kg.

At the time of assessment, the patient complained of upper gastrointestinal tenderness and constipation, accompanied by persistent nausea and vomiting, with dietary history revealing a markedly reduced oral intake at home consisting of only thin soups, mashed vegetables, and occasional reconstituted powdered nutritional supplements which were poorly tolerated. The patient's medications, including those for symptom management, are summarised by class in Table 1.

Using the Nutritional Risk Screening 2002 (NRS-2002) tool which evaluates nutritional risk by considering recent weight loss, reduced food intake, BMI, and disease severity, the patient scored a 5, indicating severe nutritional risk.[9] The Global Leadership Initiative on Malnutrition (GLIM) criteria were subsequently applied, which combines phenotypic measures (weight loss, low BMI, reduced muscle mass) with aetiological factors (reduced food intake or assimilation, inflammation) to then establish a diagnosis and grading of malnutrition.[10] Based on GLIM, the patient was classified as having severe malnutrition.

Table 1: Patient medications and classes

Medication	Class
Thiamine 200 mg IV BD	Vitamin B1
Paracetamol 1 g PO QID	Analgesic
Tramadol 50 mg PO TDS	Proton pump inhibitor
Clexane 40 mg SC OD	Opioid analgesic
Ondansetron 4 mg IV TDS	Anticoagulant
Lactulose 10 ml PO TDS	Antiemetic
Pantoloc 40 mg IV BD	Osmotic laxative

IV: intravenous; PO: by mouth; SC: subcutaneous; BD: twice daily ; TDS: three times daily; QID: four times daily; OD: once daily; mg: milligrams; g: grams.

According to the European Society for Clinical Nutrition and Metabolism (ESPEN) guidelines, this patient was also identified as high nutritional risk, defined by the presence of any one of the following: unintentional weight loss > 10–15% within the past six months, BMI < 18.5 kg/m², Subjective Global Assessment (SGA) Grade C, or preoperative serum albumin < 30 g/l (in the absence of hepatic or renal dysfunction).[8] ESPEN recommends that these patients should receive preoperative nutritional intervention to improve surgical outcomes and reduce postoperative complications.[8]

Lastly, the patient met the National Institute for Health and Care Excellence (NICE) criteria for being at high risk of refeeding syndrome as outlined in Table 2.[11]

Initial laboratory investigations are displayed in Table 3 and revealed a normal glycated haemoglobin (HbA1c) despite a prior diagnosis of Type 2 diabetes mellitus. A mildly elevated urea and creatinine along with a reduced eGFR suggested renal impairment, likely pre-renal in nature due to dehydration from persistent vomiting and inadequate oral intake. Serum albumin was within the normal range (38 g/l); however, given the patient's dehydration, this value may not be accurate.

Nutritional management

Given the patient's poor tolerance of oral intake secondary to his bowel obstruction, parenteral nutrition (PN) was initiated on May 8, 2025 following a two-day trial of oral nutritional supplements (300 kcal, 20 g protein per unit), of which the patient only managed two per day. The decision was further supported by the plan for surgical intervention, in line with the ESPEN guidelines recommending PN for patients who are malnourished or are at risk of malnutrition when oral or enteral nutrition is insufficient in the perioperative period.[8] Thiamine 200 mg was administered intravenously prior to parenteral feeding as per the NICE recommendations for refeeding syndrome, and serum electrolytes were monitored daily (Tables 1 and 3).[11] PN was delivered using a three chamber bag with vitamins and trace elements being provided separately. Feeding was commenced at a rate of 22 ml/hour over 24 hours, providing approximately 10 kcal/kg and 0.4 g protein equivalents (Eq) per kg, and was advanced daily by 5 kcal/kg/day as electrolytes remained stable, reaching a final rate of 62 ml/hour on day 6, providing 28 kcal/kg and 1.2 g protein Eq/kg. Oral nutrition support was continued concurrently providing a total of 38 kcal/kg and 1.75 g protein/kg per day; however, these were not always tolerated, and the effective absorption of these supplements could not be guaranteed. Free fluids were taken orally as tolerated by the patient for comfort.

Table 2: National Institute for Health and Care Excellence (NICE) criteria for identifying patients at high risk of refeeding syndrome[11]

High risk if ONE or more of the following:	OR	High risk if TWO or more of the following:
BMI <16 kg/m²		BMI <18.5 kg/m²
Unintentional weight loss >15% in last 3–6 months		Unintentional weight loss >10% in last 3–6 months
Little or no nutritional intake for >10 days		Little or no nutritional intake for >5 days
Low potassium, phosphate, or magnesium prior to feeding		History of alcohol abuse or use of drugs such as insulin, chemotherapy, antacids, or diuretics

ESPEN guidelines were used to guide macronutrient targets.[8,12] The patient's energy requirements were estimated at 25–30 kcal/kg/day, and protein targets ranged from 1.0–1.5 g/kg/day. Glucose infusion rates remained below 4 mg/kg/min, and lipid provision was limited to ≤ 1 g/kg/day.

Preoperative monitoring

ESPEN guidelines recommend preoperative nutritional support for 7–14 days for patients with severe nutritional risk, even if this necessitates delaying surgery, as it has been shown to optimise nutritional status, enhance metabolic resilience, and reduce postoperative complications, thereby improving surgical outcomes.[8,13] In this patient, nutritional management was initiated promptly and continued for 14 days prior to surgery, allowing for monitoring of biochemistry, weight, and body composition changes.

The patient's admission weight was 63.3 kg. By day 8 of nutritional management (day –7 relative to surgery), his weight had increased to 70.8 kg with the rapid gain most likely attributable to fluid accumulation rather than true tissue accretion. Bioelectrical impedance analysis (BIA), first conducted on this day, demonstrated a low whole-body phase angle of 3.6°, consistent with impaired cellular integrity and function, together with an elevated extracellular-to-intracellular water (ECW/ICW) ratio, supporting the interpretation of fluid overload. By day 14 of management (day –1 relative to surgery), his weight had decreased to 68.2 kg, a reduction consistent with fluid redistribution reflected by a lower ECW/ICW ratio and an improved phase angle of 4.1°, suggesting recovery of cell membrane integrity, hydration, and nutritional status (Figures 1 and 2).

Lastly, skeletal muscle mass estimates derived from BIA declined from 32.4 kg on day 8 to 31.1 kg on day 14; however, this apparent reduction is again likely a consequence of changes in hydration status as an elevated ECW/ICW ratio at the first measurement may have led to an overestimation of muscle mass, with the subsequent decrease reflecting improved fluid balance rather than true muscle loss (see Figure 1).

While functional measures such as handgrip strength were not assessed, BIA offered valuable insight into the patient's response to nutrition therapy preoperatively.

The patient's biochemistry during this period remained largely unremarkable, as shown in Table 3, with no electrolyte abnormalities or hepatic dysfunction observed, despite receiving parenteral nutrition and being at high risk of refeeding syndrome. Of note, his blood glucose also remained within normal ranges.

Table 3: Serial laboratory investigations for the patient throughout admission.

			Day of nutritional management										
Test	**Unit**	**Reference**	**1**	**3**	**4**	**5**	**6**	**9**	**13**	**16**	**17**	**19**	**20**
Sodium	mmol/l	136–145	145	143	138	143	139	138	141	**131**	136	139	141
Potassium	mmol/l	3.5–5.1	4.1	4.0	3.3	4.3	4.1	4.4	5.1	4.3	3.6	4.3	4.7
Chloride	mmol/l	98–107	101	–	–	–	–	–	–	–	–	–	–
Urea	mmol/l	2.1–7.1	**9.5**	7.1	6.5	6.4	6.7	6.1	6.3	**8.1**	6.8	4.3	3.8
Creatinine	mmol/l	64–104	**131**	103	93	86	85	82	90	97	99	72	75
eGFR (CKD-EPI)	ml/min	> 60	**53**	70	79	87	89	92	83	75	74	100	98
Calcium	mmol/l	2.15–2.50	2.26	2.36	**2.13**	**2.12**	**2.08**	2.22	2.26	–	–	–	**2.06**
Magnesium	mmol/l	0.63–1.05	0.87	0.82	0.77	0.82	0.76	0.81	0.88	–	–	–	0.81
Phosphate	mmol/l	0.78–1.42	1.36	1.35	**1.44**	1.32	1.11	1.42	**1.61**	–	–	–	1.31
Albumin	g/l	35–52	38	–	–	–	**28**	**31**	**31**	**23**	–	**26**	**28**
Total Bilirubin	µmol/l	5–21	15	–	–	–	**4**	5	5	7	–	**3**	5
Conjugated bilirubin	µmol/l	0–3	**8**	–	–	–	3	3	3	**4**	–	2	3
ALT	U/l	10–40	24	–	–	–	12	11	12	22	–	12	12
AST	U/l	15–40	27	–	–	–	13	13	13	24	–	8	11
ALP	U/l	53–128	68	–	–	–	**48**	64	82	76	–	81	91
GGT	U/l	< 40	24	–	–	–	17	30	49	39	–	34	40
LDH	U/l	100–190	–	–	–	–	–	–	–	186	–	–	–
HbA1c	%	< 5.7	4.5	–	–	–	–	–	–	–	–	–	–
CRP	mg/l	< 10	–	–	–	–	1	8	–	–	–	**65**	**44**

Units of measure: mmol/l: millimoles per litre; ml/min: millilitres per minute; g/dl: grams per decilitre; L/l: litres per litre; µmol/l: micromoles per litre; pg: picograms; fL: femtolitre; U/l: units per litre; mg/l: milligrams per litre.
Renal function: eGFR: estimated glomerular filtration rate.
Liver function tests (LFTs): ALT: alanine aminotransferase; AST: aspartate aminotransferase; ALP: alkaline phosphatase; GGT: gamma-glutamyl transferase.
Additional markers: HbA1c: glycated haemoglobin; CRP: C-reactive protein; LDH: lactate dehydrogenase.
The values shown in bold are abnormal values (too high or too low)

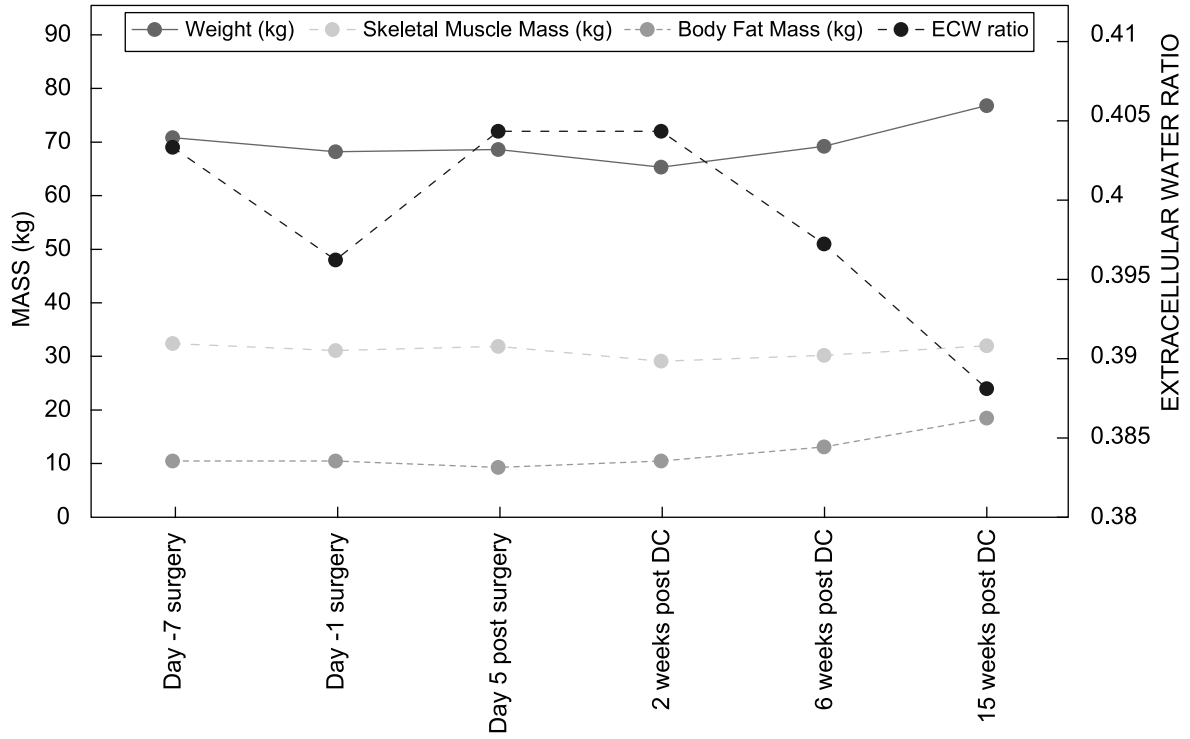

Figure 1: Longitudinal trends in body composition and extracellular water ratio across the perioperative and post-discharge period. DC: discharge.

Surgery and postoperative nutrition

The patient underwent a small bowel resection and right hemicolectomy with an ileocolic anastomosis on May 21, 2025. Preoperative octreotide was administered to reduce the risk of carcinoid crisis, although the patient had no clinical evidence of carcinoid syndrome and normal urinary 5-HIAA levels. Histology of the resected bowel mass found 25 cm from the ileocecal valve confirmed a well-differentiated neuroendocrine tumour (World Health Organization [WHO] grade 1) infiltrating the small bowel and mesenteric tissue with the involvement of multiple lymph nodes and a small peritoneal nodule. An intraoperative liver biopsy revealed only a biliary adenoma, with no evidence of hepatic metastases.

Postoperatively, PN was continued for 48 hours at the same rate of 62 ml/hour providing 25 kcal/kg and 1 g protein Eq on his last weight of 68.2 kg, while oral intake was reintroduced cautiously starting with liquids. A soft diet was introduced on postoperative day 2, alongside two oral nutritional supplements providing a total of 600 kcal and 40 g protein per day. Concurrently, PN

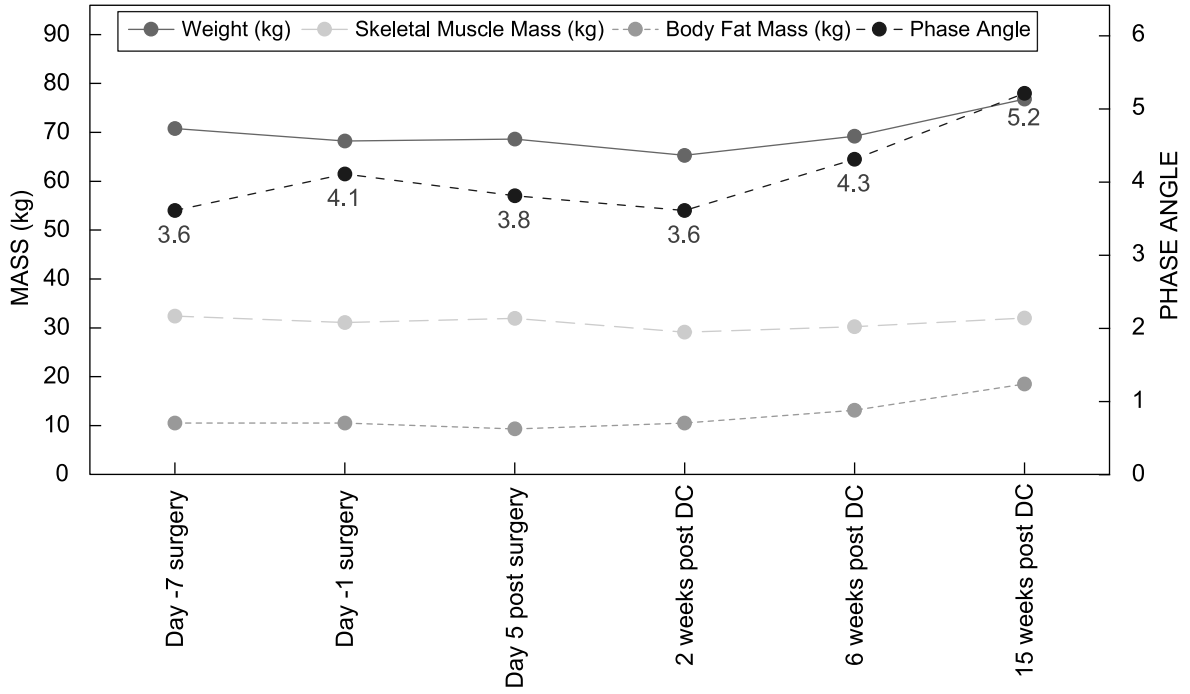

Figure 2: Longitudinal trends in body composition and phase angle across the perioperative and post-discharge period. DC: discharge

was reduced to 52 ml/hour, delivering approximately 29 kcal/kg and 1.4 g protein/kg combined with the supplements. PN was discontinued on postoperative day 4 following the surgeon's decision over the weekend, despite the patient's oral intake remaining below 60% of estimated requirements due to early satiety. The patient otherwise tolerated oral intake well, with no nausea, abdominal cramps, or vomiting being reported. He had also passed a normal stool by postoperative day 5, indicating the resumption of normal bowel function.

Discharge and follow-up

On postoperative day 5, the patient was discharged in a stable condition. His weight was 68.8 kg, and he was tolerating only one oral nutritional supplement per day in addition to approximately 50% of hospital meals. Packets of powdered nutritional supplements were issued to the patient with a daily prescription of two servings of a high-protein drink (238 kcal and 9 g protein per serving) and one serving of a fortified porridge (188 kcal and 7.1 g protein) to be taken at home between meals.

At his first outpatient review on June 10, 2025, the patient's weight had dropped by 3.5 kg in 2 weeks although he reported an improvement in appetite, no further nausea or vomiting, and regular bowel movements. He was consuming food more confidently but was only taking the prescribed nutrition therapy products twice daily, and not three times as indicated. His total body water decreased by 3.6 L, which may account for the large drop in total body weight whilst his ECW/ICW ratio remained the same.

The second outpatient visit was scheduled for 6 weeks after his operation. In the interim, the patient was advised to take the high-protein drink and fortified porridge as initially prescribed and was counselled on food fortification strategies that were to be implemented at home. At his next review, he had gained 3.9 kg in total weight, with 1 kg being skeletal muscle mass, alongside improvements in both ECW/ICW ratio and phase angle, suggesting an improvement in overall nutritional status. Supplementation was then adjusted to once-daily fortified porridge, with an emphasis on including more protein at each meal and increasing physical activity to aid in muscle gain.

At the final follow-up two months later, his weight had gone up to 76.8 kg, with only a 1.8 kg increase in skeletal muscle mass and the remainder reflecting fat gain. Despite this, the patient's ECW/ICW ratio and his whole body phase angle (now 5.2°) did improve further. These outpatient body composition changes are presented in Figures 1 and 2.

The patient was subsequently discharged from the outpatient nutrition programme and dietetic follow-up on this day, receiving advice on general healthy eating practices, including sufficient protein at each meal and increasing physical activity. An oncology consultation is still pending for the planning of potential adjuvant chemotherapy, and the patient may be re-referred to dietetic services should nutritional issues arise again.

Conclusion

Malnutrition in small bowel neuroendocrine tumours is multifactorial and significantly impacts recovery. In this case, early, targeted nutritional interventions led by the dietitian incorporating preoperative nutrition assessment and structured pre- and postoperative nutrition support facilitated improvements in fluid balance, muscle preservation, and overall nutritional status in this patient. These findings highlight the importance of proactive nutritional management to optimise surgical outcomes and long-term recovery in patients with NETs. Given the limited data on NETs and its nutritional management in Africa and South Africa in particular, further research is needed in this population to inform local practice and improve patient outcomes.

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