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CASE REPORT

Managing the complications of a complicated upper gastrointestinal surgery

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Introduction

A 74-year-old male diagnosed with adenocarcinoma of the distal oesophagus was referred to the dietetics team for post-operative nutrition support in the critical care unit (ICU). The patient was scheduled to undergo an oesophagectomy with gastric pull-up. He completed a full course of neo-adjuvant chemotherapy four weeks prior to surgery. Comorbidities included chronic obstructive pulmonary disease (COPD) secondary to cigarette smoking, uncontrolled diabetes mellitus, and hypertension.

Oesophageal cancer is the eighth most common cancer worldwide and the sixth most common cause of death due to malignancy.[1–3] Two major pathological subtypes exist: squamous cell carcinoma (SCC) and adenocarcinoma (AC). SCC accounts for approximately 90% of cases of oesophageal cancer globally; however, the incidence of AC is rising rapidly in Europe and North America.[4]

Heavy alcohol consumption, smoking, and their combined effects are major risk factors for oesophageal SCC in Western populations, while malnutrition, nitrosamines, and the human papilloma virus are risk factors in Africa and Asia. Obesity and gastro-oesophageal reflux disease with resultant oesophageal intestinal metaplasia are the main risk factors in development of AC of the oesophagus.[3,4]

The symptoms of oesophageal cancer include progressive dysphagia, gastrointestinal bleeding, recurrent aspiration or emesis, and unexplained weight loss.[2,4] Diagnosis hinges on upper gastrointestinal endoscopy and histological confirmation.[1,4] Initial choice for treatment is based on clinical staging and risk assessment, which includes a comprehensive physical examination, endoscopy, and contrast-enhanced computed tomography (CT) scan or fluorodeoxyglucose-positron emission tomography (FDG-PET) scan of the chest and abdomen.[4] Curative treatment includes preoperative chemoradiation or perioperative chemotherapy and surgical resection.[1,4]

Patients with oesophageal carcinoma are at very high risk of malnutrition and significant weight loss. More than 40% of patients lose > 10% of their bodyweight prior to surgery. This is as a result of the physical and metabolic effects of the malignancy itself, as well as the side effects of the oncological treatments. Weight loss confers an increased operative risk, worsens quality of life outcomes and is associated with poor survival in advanced disease.[2,3,5,6]

Nutritional support is an integral part of medical management in the perioperative and palliative care of these patients.[2,6] Post-oesophagectomy, the feeding route chosen remains the preference of the surgeon, as there is no evidence of advantage of one route over another. Enteral feeding administered via a nasojejunal tube (NJT) may be considered less invasive than a surgically placed feeding tube. Enteral nutrition may decrease pulmonary complications and anastomotic leak rates, maintain a higher albumin level and possibly improve nutritional status when compared with parenteral nutrition (PN). However, the most common complication associated with NJT is displacement, which occurs in 20–35% of patients and results in interruption of feed administration while awaiting replacement. This then results in delayed nutrition and increased risk of malnutrition.[7,8] Other complications include tube occlusion, gastrointestinal complaints, and patient discomfort (e.g. painful throat, nasal discomfort, tube reinsertion).[8]

Malnutrition screening and assessment

The patient was screened according to the Malnutrition Universal Screening Tool (MUST).[9] The patient's weight was 63 kg, his height was 1.7 m, and body mass index (BMI) 21.7 kg/m^2. There was significant weight loss of 7 kg (10%) from his usual weight of 70 kg in less than six months. The MUST total score was 1 and classified him as medium risk.

A full nutritional assessment was subsequently conducted. The biochemical values on admission did not indicate any significant abnormalities (see Table 1). Clinically, the patient presented with weight and lean body mass loss. The patient's dietary history indicated that he had progressive dysphagia to solids, but he did not report a major reduction in the quantity of oral intake prior to his admission. It was suspected that he was underreporting the extent of his decreased intake. According to the Global Leadership Initiative on Malnutrition (GLIM) criteria, a patient is diagnosed as malnourished if there are both phenotypic and aetiologic criteria present.[10] This patient presented with 10% weight loss (phenotypic criteria) and two aetiologic criteria, a gastrointestinal (GIT) condition that adversely impacts food assimilation as well as chronic disease-related inflammation. He was diagnosed as moderately malnourished based on the phenotypic criteria of 10% weight loss occurring in six months.[10]

Table 1: Patient biochemical values on admission

Biochemical parameter	Normal range	Value (on admission)
Sodium (Na)	135–145 mmol/L	136
Potassium (K)	3.5–5.2 mmol/L	4.0
Chloride (Cl)	96–106 mmol/L	104
Urea	6–24 mg/dL	6.2
Creatinine	59–104 µmol/L	82
Estimated glomerular filtration rate (eGfr)	≥90 mL/min/1.73 m^2	81
Haemoglobin	14.3–18.3 g/dL	14.2

Medical management

Chemoradiation together with oesophagectomy is used in the management of resectable oesophageal cancer.[2,4] Complications of oesophagectomy include pulmonary complications, anastomotic leak, and, less frequently, chylothorax and recurrent laryngeal nerve injury.[2,7] Recurrent laryngeal nerve damage with vocal cord paralysis may result in aspiration and recurrent pneumonias. Functional disorders such as gastric emptying disorders (accelerated or delayed emptying), gastroesophageal reflux, anastomotic strictures, and dumping syndrome are common in the long term.[2,9] Postoperative complications as well as the cumulative effect of multiple complications can significantly impact patient survival.[9]

The long-term effects on the nutritional status of patients post oesophagectomy include persistent weight loss and malnutrition, reduced quality of life and impaired immune function. Weight loss can be severe (> 15% of preoperative weight in 33.8% of patients after three years, and 36% of patients after five years). In the first postoperative year frequently reported symptoms include early satiety, postprandial dumping, reflux of food and/or fluids, and the absence of hunger. One year after surgery most patients still eat smaller and more frequent meals, experience an altered stool frequency, and experience a loss of enjoyment in the social aspects of eating.[1–3]

This patient underwent a hybrid (both laparoscopic and open) three-stage (McKeown) oesophagectomy, gastric pull-up, and vagotomy. A pyloroplasty was not done. Post surgery the patient was admitted to the ICU fully ventilated and on 0.34 µg/kg/minute of noradrenaline with a lactate of 11 mmol/L. The patient required postoperative ventilation because of his underlying COPD, prolonged one-lung ventilation intraoperatively and poor PaO_2/FiO_2 ratios postoperatively. Furthermore, the patient was haemodynamically unstable on high-dose vasopressors with a significant metabolic acidosis.

The following day, metabolic acidosis had resolved, the lactate levels decreased to below 2 mmol/L, and total parenteral nutrition (TPN) via a central venous catheter (CVC) was requested as an enteral feeding tube was not placed intraoperatively.

Due to the permanent anatomical change, short- and long-term nutritional goals need to be established. Early enteral nutrition is recommended via tube feeding, by a surgically placed jejunostomy, a nasoduodenal or jejunal tube, or via oral intake as it reduces the number of complications and length of hospital stay. In many cases during the first few days postoperatively oral intake is contraindicated due to the risk of an anastomotic leak and aspiration, but this is case specific.[2,6]

Initial nutrition care plan

The European Society for Clinical Nutrition and Metabolism (ESPEN) guidelines for ICU patients recommend, when using predictive equations, implementing hypocaloric nutrition (not exceeding 70% of estimated requirements) for the first week of ICU stay. The energy requirements were estimated using the predictive equation 20–25 kcal/kg/day.[11] The 2009 ESPEN guidelines on Parenteral Nutrition for Surgery recommend 25 kcal/kg/day.[12] The decision was made to initially follow the American Society for Parenteral and Enteral Nutrition (ASPEN) refeeding syndrome (RS) guidelines and then to increase the energy administration to 25 kcal/kg/day at the end of the first week of ICU stay. Regarding protein administration, the ESPEN critical care guidelines recommend 1.3 g/kg/day from day 3 onwards and the Parenteral Nutrition for Surgery recommends 1.5 g/kg/day. The goal was set at 1.3 g/kg/day and to monitor the patient and increase as needed due to the moderate malnutrition present.[11,12]

Complications

Throughout the treatment of the patient, he experienced several complications that had an impact on nutritional management. The complications are grouped for the ease of discussion into refeeding syndrome, enteral feeding intolerance, and, lastly, parenteral nutrition-associated liver disease (PNALD). Figure 1 gives an overview of the complication's timeline.

Complication 1: refeeding syndrome

Refeeding syndrome (RS) refers to a range of metabolic and electrolyte alterations after the reintroduction and/or increased provision of energy after a period of decreased or no energy intake.

Patients with malignancy can be at risk of RS due to prolonged starvation and/or electrolyte losses.[13] Additionally, this patient was diagnosed as moderately malnourished according to the GLIM criteria and thought to be at risk of developing RS.[10] Unfortunately, the available screening strategies (including the GLIM criteria) for identifying patients at risk of RS are

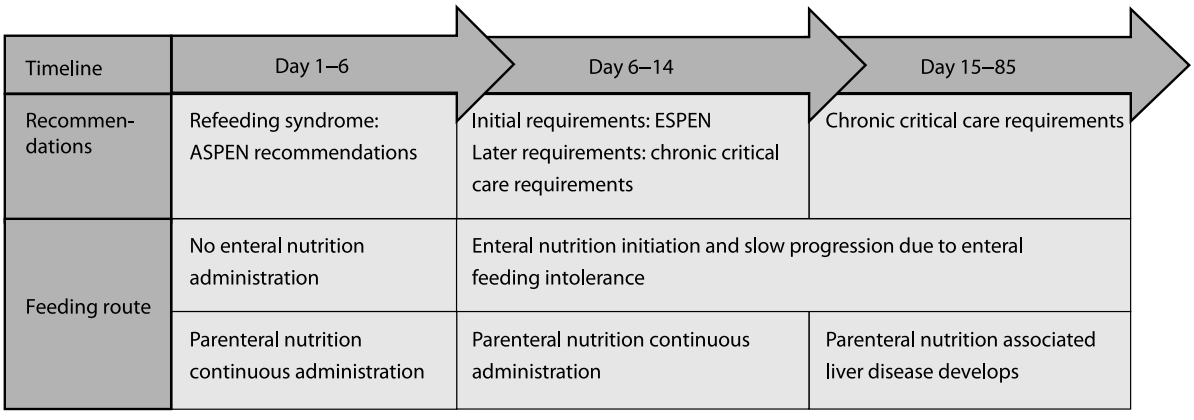

Figure 1: Nutritional journey of patient. ASPEN: American Society for Parenteral and Enteral Nutrition, ESPEN: European Society for Clinical Nutrition and Metabolism.

imprecise and poorly validated. This is exacerbated by the lack of a consensus definition for RS.[13]

With extended periods of nutrition deprivation, insulin production is decreased and glucagon production increased, resulting in increased glycogenolysis. As nutrition deprivation continues, gluconeogenesis occurs with resulting increased protein catabolism. With prolonged fasting, lipolysis occurs, and the body's basal metabolic rate decreases by 20–25%. During this time, potassium, phosphate, and magnesium become severely depleted. However, the serum levels may remain in normal ranges due to decreased renal excretion and phosphate moving from the intracellular level into the blood.[13–15]

Upon the introduction of carbohydrates, insulin secretion increases as metabolism shifts from fat to carbohydrates. Insulin release stimulates anabolic processes that require minerals such as an increased intracellular demand for phosphate due to phosphorylation of glucose as glycolysis is initiated and the resulting increased adenosine triphosphate (ATP) formation. There is a shift of potassium intracellularly due to the sodium potassium ATPase (Na-K-ATPase) symporter, a cell-wall enzyme that is responsible for the flow of glucose and potassium into and sodium out of the cell. Magnesium is an important cofactor for phosphorylation of ATP and hypomagnesaemia also impairs potassium reuptake in the nephron, resulting in increased losses of K. It may also impair cellular transport of potassium as the Na-K-ATPase is magnesium dependent. Thiamine deficiencies may also occur during RS as it is a cofactor for glucose-dependent metabolic pathways and plays a role in the conversion of lactate to pyruvate. All these electrolyte shifts, along with the already depleted mineral pool, could result in profoundly decreased levels of PO_4, Mg, and K.[14,15]

The ASPEN guideline for preventing RS is to initiate energy with 10–20 kcal/kg for the first 24 hours (see Table 2 for the ASPEN consensus guidelines on RS).[13] This patient's initial dietary prescription was calculated at 10 kcal/kg/day on Day 1. There is no recommendation for protein administration, and it depends on the advancement of energy administration and nutritional product selection. Parenteral nutrition was requested on the day of admission to the ICU and a three-chamber PN bag was prescribed at 50 mL/hour/24 hours. It provided 800 kcal/day (12.7 kcal/kg/day) and 38 g/day (0.6 g/kg/day) protein. The energy was slightly above the calculated requirements but still within the recommendations. Intravenous thiamine (200 mg/day),[15] a multivitamin providing both soluble and insoluble vitamins,[13] and trace elements were also prescribed. However, the pharmacy did not have the prescribed bag in stock and consulted another member of the medical team on an alternative bag to be issued. This bag was initiated at the same rate as the previously prescribed TPN and provided 21 kcal/kg/day (double the amount of energy prescribed) and 1.2 g/kg/day of protein. The following day (Day 2) there was a slight drop in magnesium levels and the treating dietitian selected an all-in-one PN bag that provided 18 kcal/kg/day and 1.0 g protein/kg/day. The intensivist prescribed 2 g of IV magnesium sulphate twice daily to address the decrease in magnesium levels.

The patient developed severe refeeding syndrome as evident by the 50% decrease in serum PO_4 levels (Table 2) on day 3 in ICU.[13] It should be noted that RS does not necessarily develop immediately the day after initiating feeds but can develop up to 48–72 hours after initiation.[13] Thus in retrospect, the energy administration should already have been decreased on Day 2 to prevent RS and not only on Day 3 to 10 kcal/kg/day so as to treat the severe RS. This decrease is in line with the

Table 2: ASPEN Consensus Recommendations for Avoidance and Treatment of Refeeding Syndrome (RS) in at-risk adult patients[13]

Aspect of care	Recommendations
Initiation of calories	• First 24 hours: start with 100–150 g of dextrose (enteral or IV) or 10–20 kcal/kg • Advance: 33% of goal every 1–2 days • Moderate to high risk of RS with low electrolyte levels: hold initiation or increase of calories until electrolytes are supplemented and/or normalised • Consider calories from IV dextrose solutions and medications infused in dextrose • If patient has received significant amounts of dextrose for several days, from maintenance IV fluids and/or medications in dextrose, and has been asymptomatic with stable electrolytes, calories from nutrition may be reintroduced at a higher amount
Fluid restriction Sodium restriction Protein restriction Electrolytes	• No recommendations • Check serum potassium, magnesium, and phosphate before initiation of nutrition • Monitor every 12 hours for the first 3 days in high-risk patients • Normal pre-feeding electrolytes: no recommendation on prophylactic dosing • Low electrolytes pre-feeding: replace based on established standards of care • Sharp electrolyte drop/difficulty correcting: decrease calories/grams of dextrose by 50% and advance the dextrose/calories by approximately 33% of goal every 1–2 days based on clinical presentation • Consider cessation of nutrition support when electrolyte levels severely and/or life-threateningly low or dropping precipitously
Thiamine and multivitamins (MVT)	• Supplement thiamine: 100 mg before feeding or initiating dextrose-containing IV fluid • Supplement thiamine 100 mg/day for 5–7 days or longer in patients with starvation, chronic alcoholism, or other risk factors for deficiency and/or signs of thiamine deficiency • PN: add multivitamin therapy (MVT) daily, unless contraindicated • Oral/enteral nutrition: add complete oral/enteral MVT once daily for 10 days or greater based on clinical status and mode of therapy

ASPEN: American Society for Parenteral and Enteral Nutrition, IV: intravenous, RS = Refeeding syndrome

ASPEN guideline that recommends decreasing energy administration with 50% when there is a severe drop in electrolyte levels.[13] To address the hypophosphatemia, intravenous (IV) potassium phosphate (KPO_4) at a dose of 20 mmol twice daily was administered. Feeds were slowly progressed over several days (see Table 3) to meet target requirements.

The protein administration over the first three days was below 1 g/kg/day and the ESPEN critical care guidelines recommend 1.3 g/kg/day.[11] As the patient was diagnosed as moderately malnourished and due to the slower progression in feeds as part of the RS treatment, the decision was made to add an intravenous amino acid modular on day 4. The modular selected provided 57 g amino acids per 500 mL of which 45% (25.6 g) is essential amino acids. The product was administered at 10 mL/hour/24 hours providing 28 g amino acids in addition to the amino acids from the PN.

Complication 2: enteral feeding intolerance

On Day 5 post-surgery, the patient was extubated. There was minimal drainage from the neck drain. On day 6 the speech therapist (ST) did a bedside swallow assessment and oral intake was found to be unsafe. The surgeon requested a barium swallow that confirmed the unsafe swallowing due to pooling of contrast and immediate aspiration. The patient was taken to theatre and a nasogastric tube (NGT) placed. The patient was started on a nutritionally complete, 1.2 kcal/mL whey peptide-based tube feed, high in protein and with 6.4 g of fibre per 1 L at 10 mL/hour/24 hours. The chosen feed was high in MCT and contained 3 g of EPA (eicosapentaenoic acid) and DHA (docosahexaenoic acid) per litre and antioxidants to induce immune-enhancing effects and to meet the stress-induced elevated needs of critically ill patients. The nutrition prescription had been advanced during this period to meet 25 kcal/kg/day energy and 1.3 g/kg/day protein. Supplemental cyclic PN was prescribed to meet these requirements and was administered for 14 hours per day with a rest period of 10 hours.

The patient experienced no nausea or vomiting but did complain of heartburn. He was started on intravenous metoclopramide and pantoprazole.[8] The feed was also changed to a less nutrient dense whey peptide-based tube feed without fibre to

Table 3: Initial biochemical parameters and nutrient administration progression

Biochemical parameter	Normal range	Day 1	Day 2	Day 3	Day 4	Day 5	Day 6	Day 7
Potassium (K)	3.5–5.2 mmol/L	4.0	4.2	4.1	4.4	3.7	3.5	3.6
Magnesium (Mg)	0.66–1.07 mmol/L	–	0.63	0.73	0.74	0.70	0.84	0.97
Phosphate (PO_4)	0.78–1.42 mmol/L	–	1.16	0.58	0.94	1.02	1.24	0.96
Energy	Kcal/kg/day	12 (21)	18	10	15	20	23	25
Protein	g/kg/day	0.6 (1.2)	1.0	0.5	0.9	1.3	1.4	1.4

Table 4: Parenteral nutrition journey of the patient

Day of ICU stay	Lipid composition	Supplemental or complete	Infusion	Energy and protein provision	NPE ratio:, glucose:fat ratio	PN: glucose oxidation rate (mg/kg/hour)	PN: fish oil dose (g/kg/day)
1	80% olive, 20% soybean oil	Complete	Continuous	21 kcal/kg/day 1.2 g/kg/day	52%:48%	1.8	0
2	SMOF	Complete	Continuous	18 kcal/kg/day 1.0 g/kg/day	44.5%:50% *see footnote	1.1	0.12
3–16	Soybean, fish oil, MCT	Complete	Continuous, gradual increase to maximum rate	10–26 kcal/kg/day 0.5–1.5 g/kg/day	60%:40%	1.0–2.9	0.04–0.12
17–27	SMOF	Supplemental	Cyclic for 18 hours	18 kcal/kg/day 1.1 g/kg/day Including EN: 25 kcal/kg/day 1.4 g/kg/day	67%:30% *see footnote	2.6–3.5	0.08–0.11
28–30	SMOF	Complete	Continuous	27 kcal/kg/day 1.25 g/kg/day	67%:30% *see footnote	2.7	0.12
31–40	SMOF	Complete	Cyclic for 18 hours	27 kcal/kg/day 1.25 g/kg/day	67%:30% *see footnote	3.7	0.12
41–42	Nil administration to rest liver						
43–91	SMOF	Supplemental	Cyclic for 10 hours	20 kcal/kg/day 0.9 g/kg/day Including EN: 31 kcal/kg/day 1.5 g/kg/day	44.5%:50% *see footnote	2.1	0.1
92–93	Soybean, fish oil, MCT	Supplemental	Cyclic for 15 hours	10 kcal/kg/day 0.5 g/kg/day Including EN: 25 kcal/kg/day 1.2 g/kg/day	60%:40%	1.6	0.04

MCT: medium chain triglycerides, SMOF: 30% soybean oil, 30% MCT oil, 25% olive oil, 15% fish oil, NPE: non-protein energy.
*Outstanding percentage (to make up total of 100% NPE) = 50 kcal from glycerol backbone found in SMOF.

monitor whether the fibre (albeit low in the initial feed) could be contributing to the intolerance. The feeds were also administered from 06:00 to 20:00, and held for the evening. During feeding hours, the patient was instructed to keep the head of bed at 30–45°. The patient reported an improvement in reflux symptoms.

On Day 16 post-surgery, the NGT was clamped and oral intake with fluids was commenced but the patient showed signs of immediate aspiration. Oral intake was stopped immediately, trophic enteral feeds via the NGT were restarted, and PN administration continued. The patient continued to show signs of aspiration and three weeks post-surgery (Day 21) the patient went for a video fluoroscopic swallowing exam (VFSE) that confirmed immediate aspiration and pooling of contrast in the hypopharynx. This is most likely a result of anatomical distortion at the site of the proximal gastric anastomoses. He was continued on TPN and kept nil per os (NPO) as he again pulled out his NGT.

A week later (Day 29) it was suspected that the patient was experiencing delayed gastric emptying along with pyloric sphincter dysfunction resulting in aspiration that necessitated re-intubation and ventilation. Pylorus-directed interventions include botulinum toxin injections, pneumatic balloon dilation, stenting, surgical pyloroplasty, and a gastric per-oral endoscopic myotomy, used to treat selected patients with gastroparesis who are unresponsive to medications.[14] The surgeon decided to place a pyloric stent for the patient in theatre. At the same time, an NJT and draining NGT were inserted. During the procedure the surgeon aspirated 750 mL gastric fluid.

Postoperatively, enteral feeds were administered via NJT. In the following two weeks the patient continued to vomit numerous times and experienced large NG drainage. On Day 50, the enteral feeds were placed on hold as NJT displacement was suspected but placement was confirmed, and dextrose water was started and progressed to a semi-elemental feed. During this period cyclic TPN was administered for 16 hours per day to meet the patient's nutritional requirements. The patient continued to experience large nasogastric drainage with episodes of vomiting, and two months after the original surgery a laparotomy was performed for a gastric outlet obstruction and another stent was placed at the pyloric sphincter as the previous pyloric stent migrated. Thereafter, full enteral feeds were only reached by Day 86 due to the patient removing his NJT multiple times, the NJT migrating resulting in feeding intolerance, as well as the patient developing acalculous cholecystitis that resulted in an open cholecystectomy.

Complication 3: liver dysfunction with intra-hepatic cholestasis

TPN was administered to the patient on admission to the ICU and continued for 93 days (see Table 4).

Two weeks from TPN initiation (D14), the patient's alkaline phosphatase (ALP) and gamma-glutamyl transferase (GGT) continued to increase. The development of PNALD was queried. PNALD is defined as an elevation of the liver enzymes by 1.5 times the upper limit of the normal parameters in the absence of other causes such as viral hepatitis or drug-induced changes.[16]

There are three types of PNALD that can develop, namely steatosis, cholestasis, and gallbladder sludge or stones.[16] The risk factors for the development of PNALD include the nutrient composition of the TPN solution, long-term TPN administration, overfeeding and the development of lipogenesis, excessive carbohydrate or lipid administration, intestinal failure or resection and inability to stimulate the gastrointestinal tract, and bacterial or fungal infections such as sepsis, or small-bowel bacterial overgrowth.[16–18]

Table 5: Practices to reduce the risk of PNALD[16–20]

Practice	Mechanism
Avoid overfeeding Energy < 25–35 kcal/kg/day Protein intake of 0.8–1.5 g/kg/day IV lipid administration < 1.5 g/kg/d Glucose load < 7 mg/kg/min	↓ risk of developing steatosis caused by excess calories depositing in the liver
Administration of the recommended carbohydrate to fat ratio 70–80% carbohydrates from non-protein energy (NPE) 15–30% lipids from NPE	↓ steatosis by reducing hepatic triglyceride uptake and promoting fatty acid oxidation
Administration of oral or enteral nutrition Early initiation of oral or enteral feeds Early progression to full enteral or oral nutrition	↑ enterohepatic circulation of bile acids
Cyclical PN < 24 hours 8–12 hours	↓serum liver enzymes and conjugated bilirubin
Mixed lipid emulsion Olive oil, soybean, fish oil, MCTs Fish oil dose 0.1–0.2 g/kg/day Lipid emulsions enriched with EPA and DHA Tocopherol containing lipid emulsions	↓ inflammation

↓: reduce, ↑: increase, IV: intravenous, PN: parenteral nutrition, MCT: medium chain triglycerides, NPE: non-protein energy, EPA: eicosatetraenoic acid, DHA: docosahexaenoic acid.

Cyclic TPN was implemented for 18 hours, to rest the liver (see Table 4) and for the other 6 hours normal saline was administered IV (see Table 5 for practices to reduce PNALD risk).[16,17] However, the patient experienced hypoglycaemia during those 6 hours and the normal saline had to be changed to sustenance. A TPN bag containing a lipid profile that has been proven to reverse the effects of PNALD was selected (see Table 4).[16,17,19] The energy requirements were kept at 25 kcal/kg/day to prevent overfeeding. The glucose to fat non-protein energy ratio, glucose oxidation rate, and fish oil dose administration can be found in Table 5.

The liver function of the patient stabilised with these interventions. During this time enteral nutrition via an NJT, placed in the post ligament of Treitz position, was attempted in order to promote enterohepatic circulation of bile acids and help stabilise the liver function. However, after three months (Day 92) of cyclic TPN and trickle feeds via a NJT, the obstructive liver enzymes increased significantly (ALP 764 IU/L, GGT 441 IU/L). An ultrasound was performed and a large, distended gallbladder with a thickened wall was observed. The patient was diagnosed with acalculous cholecystitis and an open cholecystectomy had to be performed. Post cholecystectomy,

the TPN was stopped and full EN feeds via the NJT were attempted.

Conclusion

The patient had an extended ICU stay due to the numerous complications experienced and need for re-intubation on several occasions. He was never able to swallow and a feeding jejunostomy was place along with a pharyngostomy for drainage of secretions. After nine months in ICU, cancer metastasis was confirmed, and he was transferred to a step-down facility.

This case study highlights a complex case post upper gastrointestinal surgery for oesophageal cancer management. Patient complications often occur simultaneously and seldom in isolation or consecutively, necessitating critical thinking and problem-solving. The importance of communication between multidisciplinary team members is crucial to effectively manage and prevent additional complications.

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