Nutritional management of a critically injured patient

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Introduction

A 28-year-old male patient was admitted with a gunshot wound in the abdomen. The gunshot wound was on the right iliac fossa, just above the right iliac crest, with no exit wound. The patient suffers from asthma and has a history of smoking.

Case report

During examination on admission (day 0), the patient was haemodynamically unstable, but responsive to fluids. He had a distended abdomen with decreased bowel sounds and was taken to theatre for an explorative laparotomy. Complete transection of the iliac vein was found and ligation was carried out. The external iliac artery had a long, devitalised segment for which the right groin was exposed to gain distal access. Use of a synthetic graft was indicated to repair the injury. Obtaining the graft from another hospital caused a delay, resulting in prolonged ischaemic time. Eventually the graft was used and an end-to-end anastomosis performed from the external proximal iliac artery to the common femoral artery. Small bowel lacerations were also found and closed. An angiogram was carried out to confirm the patency of the superficial femoral artery. Muscles of the right leg were considered borderline in terms of viability, and fasciotomies were executed. The patient was then admitted to the intensive care unit (ICU) on inotropes and full ventilation.

On day 2, a weak, but positive, dorsalis pedis pulse could be felt and the patient was taken back to theatre for a "relook" fasciotomy. The lateral compartment muscles were necrotic and debridement was carried out. On day 3, serum levels of myoglobin and creatine kinase (CK) had increased markedly, and on days 4 and 6, haemodialysis was introduced to treat fluid overload and myoglobinuria (Table I).

On day 5, extensive debridement of the lateral compartment was performed to remove necrotic tissue. Further debridement of the proximal and distal muscles had to be performed on day 7. The patient was taken to theatre for wound inspection on day 9, as serum myoglobin and CK levels had again increased, but the muscles in all the compartments were found to be viable. The patient was extubated on day 10 and transferred to the high care unit on day 11. Eventually, an above-knee amputation had to be performed on day 14. The patient then recovered well and was transferred to the surgical ward on day 18. On day 22, he was discharged from hospital.

Diagnosis

The diagnosis was critical injury, related to an abdominal gunshot wound to the right iliac artery and prolonged ischaemic time, which eventually led to an above-knee amputation. Small bowel lacerations were closed, with no complications.

Table I: Serum myoglobin and creatine kinase levels

Day	0	1	2	3	4	5	6	8	9	12	13	14	15	18	20
S-myoglobin (16-96 µg/l)	1 451	37 160	15 383	6 420	2 878	1 529	735	320	1 093	173	319	309	1 325	338	159
S-CK (20-200 U/l)	-	-	-	20 106	13 240	7 411	3 272	1 264	3 163	512	1 048	1 986	-	-	706
Comment		PO				PD		PD					Post AKA		

AKA: above-knee amputation, PD: post dialysis, PO: postoperative

Anthropometry

Recumbent height was measured as 173 cm. The patient's left mid-arm circumference was 31.8 cm (50th percentile) and his weight was estimated at 75 kg with a body mass index value of 25 kg/m^2. This correlated well with his clinical appearance.

Nutritional management

Key considerations in the nutritional therapy of ICU patients include route of feeding (parenteral, enteral or both), when to feed (within 24-48 hours, and preferably early enteral nutrition), and what to feed (standard feeds or feeds with pharmaconutrients).[1]

From admission, the dietitian was involved in the patient's care as a multidisciplinary approach is a standard of care in the ICU in which the patient was treated. Table II provides a summary of the nutritional management of the patient, which will then be discussed in more detail.

Table II: Summary of nutritional management in the intensive care unit

Day	0	1	2	3	4	5	6	7	8	9	10	11
Intervention												
Intravenous glutamine	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓
Total parenteral nutrition	✓	✓	✓	✓	✓	✓	✓	✓	✓			
Trophic feeds (nasogastric)			✓	✓	✓	✓	✓					
Full enteral feeds								✓	✓	✓	✓	
Oral feeds												✓

Upon admission to the ICU (day 0), the dietitian requested the start of intravenous glutamine (L-alanyl-L-glutamine dipeptide) infusion at 12 ml/hour to provide 0.5 g/kg glutamine (a total of 38.8 g/day). Intravenous glutamine infusion was administered until day 11. Glutamine supplementation of parenteral nutrition is a grade A recommendation by all expert guidelines as it can improve survival in a critically ill patient.[1,2] A parenteral glutamine dose of 0.5 g/kg appears to be optimal for survival and beneficial outcomes have been reported in several studies.[1] As this patient was still on high doses of inotropes, total parenteral nutrition (TPN) was also ordered on admission to provide early nutritional support. TPN was given at 70 ml/hour, providing 14.6 g N, 1 505 kcal non-protein energy, 190 g dextrose, 76 g lipid and 10.2 g glutamine. The intravenous glutamine was then reduced to 9 ml/hour to continue glutamine administration at 0.5 g/kg. Starting energy requirements were calculated as 84 kJ/kg (20 kcal/kg) and 1.5 g/kg of protein.

As illustrated in Table II, the patient received TPN and intravenous glutamine on days 0 and 1. On day 2, the patient was haemodynamically stable and trophic feeds, containing glutamine, antioxidants and tributyrin, were started via nasogastric tube at 10 ml/hour, together with TPN, and increased to 20 ml/hour on day 4. This provided enteral glutamine at 0.38 g/kg, in addition to the intravenous glutamine of 0.5 g/kg. An enteral dose of glutamine, greater than 0.3 g/kg, is required for benefit to be achieved.[1] In the literature, it is not clear if enteral glutamine should be administered in addition to intravenous glutamine. In this case, the enteral glutamine was given in addition to the intravenous glutamine from days 2-6. Enteral nutrition has become a recommended standard of care for ICU patients who are haemodynamically stable within 24-48 hours of arrival in ICU.[1]

TPN, together with trophic feeds, was given until day 6. By day 7, enteral nutrition was started and the TPN was weaned. A high-protein, high-energy polymeric feed (75 g protein/l and 6 300 kJ/l) was used to meet the calculated nutritional requirements of the patient. The feed did not provide enteral glutamine. Therefore, enteral glutamine administration had stopped by day 7. The energy requirement was calculated using 105 kJ/kg (25 kcal/kg) and protein as 1.5 g/kg. By day 9, the patient was on full enteral nutrition at a target rate of 65 ml/hour, with continued intravenous infusion of glutamine dipeptide at 12 ml/hour (38.8 g glutamine/day, i.e. 0.5 g/kg).

On day 11, the patient was started on oral intake as he was extubated and fully awake. While in ICU, daily nutrient intake was calculated.

A question that is often asked by dietitians is whether the protein that is provided by the intravenous glutamine dipeptide should be added to the total protein prescription. General consensus is that it should not be. Table III provides a summary of the patient's daily glutamine intake, as well as protein intake, with and without the protein content of the supplemental glutamine. The total protein intake, including protein from supplemental glutamine, resulted in intakes between 2-2.2 g protein/kg. Monitoring the serum urea gives a good indication of possible protein overfeeding.

The patient's renal and liver functions were monitored throughout the course of ICU treatment (Table IV). Glutamine administration may increase blood urea nitrogen or ammonia levels in patients with hepatic or renal failure. According to Wischmeyer of the University of Colorado Health Sciences Centre,[3] they tolerate blood urea nitrogen increases up to 12.7 mmol/l (75 mg/dl) with their patients. The maximum level recorded in this patient was 9.2 mmol/l.

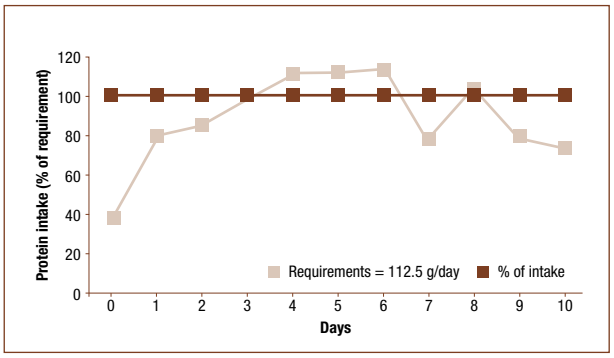
Table III: Daily glutamine and protein intake

	Day	0	1	2	3	4	5	6	7	8	9	10
Glutamine intake (g)	Intravenous	33.7	39.3	34.5	35.98	29.2	29.6	30.8	33.3	36.3	33.9	38.8
	Enteral	0	0	6	14	24.6	24	22.8	0	0	0	0
	TOTAL	33.7	39.3	40.5	50.4	53.8	50.3	50.5	33.3	36.3	33.9	38.8
Protein intake (g)	Without glutamine protein	41	90	95	110	125	127	75	89	115	88	83
	Glutamine protein added	84	133	131	149	153	151	147	124	166.5	138.2	140.5

Table IV: Biochemistry (renal and liver functions)

Day	0	1	2	3	4	5	6	7	8	9	10	11	12	Normal value
Serum urea (mmol/l)	7.6	7.1	6	7.1	6.2	6.3	7.1	7.5	7.9	9.2	8.7	8.9	7	2.6-7.0
Serum creatinine (µmol/l)	126	89	75	81	66	72	81	70	85	76	59	66	70	60-100
Total bilirubin (µmol/l)				13	20						18	12	9	0-21
Conjugated bilirubin (µmol/l)				7									5	0-6
GGT (U/L)				47							498		335	1-24
ALT (U/L)				103							183		128	5-40
AST (U/L)				354							173		112	8-20
ALP (U/L)				110							414		373	40-120

ALP: alkaline phosphatase, ALT: alanine aminotransferase, AST: aspartate aminotransferase, GGT: gamma glutamyl transferase



%: percentage

Figure 1: Daily protein intake as a percentage of the requirement

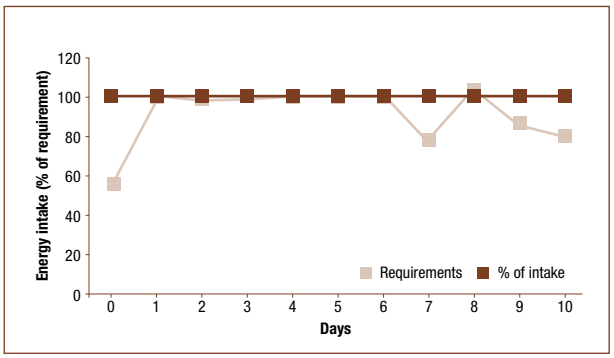


Figure 2: Daily energy intake as a percentage of the requirement

Supplying adequate energy and protein is the most fundamental form of nutritional therapy.[2] The patient's protein and energy intake are illustrated in Figure 1 and Figure 2 respectively. Despite several interruptions in feeding time because of surgical interventions, energy and protein intake could mostly be obtained between 80% and 100% of calculated requirements.

Discussion

Critical illness is associated with catabolic hormonal and cytokine responses, including increased levels of counter-regulatory hormones (cortisol, catecholamines and glucagon), increased levels of proinflammatory cytokines in blood and tissue [interleukin (IL)-1, IL-6, IL-8 and tumour necrosis factor-alpha] and peripheral tissue resistance to anabolic hormones (insulin and insulin-like growth factor 1). This hormonal milieu increases glycogenolysis and gluconeogenesis, causing a net breakdown of skeletal muscle and enhanced lipolysis to provide endogenous glucose, amino acids and free fatty acids that are required for cellular and organ functioning and wound healing.[4,5] There is an increase in protein catabolism, along with a marked increase in urinary losses of nitrogen, phosphorus, sulphur, potassium, magnesium and creatinine.[5] But, although these plasma substrate levels may be increased, their availability for use by peripheral tissues may be decreased and plasma levels of certain substrates, e.g. glutamine, may be insufficient to meet metabolic demands.[4]

Both glutamine and antioxidants have been found to play key roles in protecting against complications such as infections and mortality in surgical, trauma and critical care settings. Glutamine serves as a metabolic substrate for enterocytes and immune cells, and thus supports barrier and immune functions. Glutamine has also recently been proposed as a signalling molecule to turn on implicated genes in cell protection and immune regulation. An example of this stress-signalling function is the role of glutamine in enhancing heat-shock protein, which is essential for cellular recovery following injury, and as protection against organ failure.[1] Typically, critically ill patients have the most severe glutamine deficiency. Glutamine deficiency on admission correlates with increased mortality.[1]

Intravenous glutamine supplementation results in a uniform uptake of glutamine across the splanchic area, similar to what occurs with endogenously-produced glutamine. The elimination rate of intravenous glutamine from the plasma is fast. Uptake is also quick in the case of enteral glutamine supplementation and occurs in the upper part of the jejunum. Only a fraction of the absorbed glutamine can be recovered in the portal blood which is indicative of elimination in the gut; most likely in the enterocytes and gut immune cells. The complete uptake of enteral glutamine in the upper part of the jejunum leaves the remaining part of the gastrointestinal tract unsupported by the enteral route. Enteral glutamine supplementation has a marginal effect on plasma glutamine concentration. It is highly likely that, when enteral nutrition is not possible, a supply of parenteral glutamine becomes critical for the intestinal tract.[6]

It is recommended that ICU patients should be fed within 24-48 hours of admission, preferably via the enteral route.[1] Enteral nutrition can be contraindicated in approximately 10-15% of critically ill patients. Generally recognised indications of TPN include small bowel

resection, proximal high output fistulae and a perforated small bowel. Other conditions in which enteral nutrition may be contraindicated or not tolerated are severe diarrhoea or emesis, substantial abdominal distension, partial or complete bowel obstruction, severe gastrointestinal bleeding and severe haemodynamic instability.[4]

It has become increasingly clear that the underutilised gut can contribute significantly to the proinflammatory state of critically ill patients, thus emphasising the role of early enteral nutrition.[7] Enteral nutrition supports the functional integrity of the gut by maintaining tight junctions between the intraepithelial cells, and stimulating blood flow inducing the release of trophic endogenous agents, such as cholecystokinin, gastrin, bombesin and bile salts.[8] Evidence suggests that as little as 25-50% of energy that is supplied via the gut generally suffices to maintain at least some of the enterally fed mucosal immune morphology, such as maintaining mucosal immunoglobulin (Ig) A levels.[9] The changes seen with decreased enteral stimulation in extraintestinal mucosal immune parameters, such as decreased IgA in the respiratory tract, indicate that the gut-associated lymphoid tissue, as well as the mucosal-associated lymphoid tissue, are affected by the type and route of nutrition.[8,9] The adverse consequences of critical illness on the reported permeability changes include an increased bacterial challenge (engagement of gut-associated lymphoid tissue with enteric organisms), the risk of systemic infection and a greater likelihood of multi-organ dysfunction syndrome.[8]

The specific reasons for providing early enteral nutrition are to maintain gut integrity, modulate stress and the systemic immune response and attenuate disease severity. Additional end-points of enteral nutrition therapy include use of the gut as a conduit for the delivery of immune-modulating agents and the use of enteral formulations as an effective means of stress ulcer prophylaxis.[8]

Optimal energy homeostasis should be an important goal in ICU patients. Both excessive and insufficient energy intake can be deleterious.[10] When enteral intake alone was inadequate for patients who were expected to be mechanically ventilated for more than 72 hours, and who had BMI scores of < 25 or < 35, each additional 1 000 kcal/day or 30 g protein/day was reported to be associated with reduced mortality.[1,2] The goal of protein provision is to maximise protein synthesis in the hope of meeting or matching catabolism. Efforts to reach nitrogen balance are important, but it is unrealistic to expect a positive balance and net increases in lean body mass.[6]

The goals of nutrition support therapy have been expanded from simply providing protein and energy to pursuing broader avenues for therapeutic interventions, such as pharmaconutrition and immune modulation.[7] Glutamine has been shown to be beneficial in improving outcomes in clinical trials in a wide range of patients.[3] A multidisciplinary approach to nutrition support is essential.[7] Findings from a study by Roberts et al suggest that length of stay in the ICU is most likely to have an inverse relationship with adequacy of nutritional support, when nutritional support is started in ICU, gastrointestinal dysfunction, and time of intervention by the dietitian during ICU. Intervention by a registered dietitian within the first three days resulted in a trend towards a shorter ICU stay.[11]

References

1. Wischmeyer PE. Malnutrition in the acutely ill patient: is it more than just protein and energy? S Afr J Clin Nutr. 2011;24(3):S1-S7.
2. Wischmeyer PE. Nutritional pharmacology in surgery and critical care: "You must unlearn what you have learned". Curr Opin Anaesthesiol. 2011;24(4):381-388.
3. Wischmeyer PE. Clinical applications of L-glutamine: past, present and future. Nutr Clin Pract. 2003;18(5):377-385.
4. Ziegler TR. Parenteral nutrition in the critically ill patient. N Eng J Med. 2009;361(11):1088-1097.
5. Joseph B, Wynne JL, Dudrick SJ, Latifi R. Nutrition in trauma and critically ill patients. Eur J Trauma Emerg Surg. 2010;36:25-30.
6. Wernerman J. Glutamine supplementation. Annals of Intensive Care. 2011;1(25):1-6.
7. Miller KR, Kiraly LN, Lowen RG, et al. "CAN WE FEED?" A mnemonic to merge nutrition and intensive care assessment of the critically ill patient. J Parenter Enteral Nutr. 2011;35(5):643-659.
8. McClave SA, Martindale RG, Vanek VW, et al. Guidelines for the provision and assessment of nutrition support therapy in the adult critically ill patient: Society of Critical Care Medicine (SCCM) and American Society for Parenteral and Enteral Nutrition (ASPEN). J Parenter Enteral Nutr. 2009;33(3):277-313.
9. Sano Y, Hermsen JL, Kudsk KA. The gut as an immune organ. Clin Nutr Highlights. 2008;4(2):277.
10. Singer P, Pichard C. Parenteral nutrition is not the false route in the Intensive Care Unit. J Parenter Enteral Nutr. 2012;36(1):12-14.
11. Roberts SR, Kennerly DA, Keane D, George C. Nutrition support in the ICU: adequacy, timelines and outcomes. Crit Care Nurse. 2003;23(6):49-57.