

SASPEN Case Study

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Background information

The patient, a 79-year-old woman, was admitted to hospital on 14 August 2009 with locked jaws, generalised body pain and dyspnoea. She presented with features suggestive of tetanus, including trismus (prolonged tonic spasm of the muscles of the jaw), generalised rigidity and irregular muscle spasms. The patient had a medical history of type II diabetes mellitus and hypertension.

Patient's course

The patient was admitted to the intensive care unit (ICU) for ventilatory support. She was given tetanus immunoglobulin intramuscularly. Complications such as hypoalbuminaemia and anasarca developed as part of the disease progression. The patient also contracted influenza whilst in the ICU. A tracheostomy was performed after two weeks' intubation. The patient's clinical picture deteriorated and she developed multi-organ failure, including septic shock, encephalopathy and renal dysfunction, and hypotension.

Diagnosis

Tetanus, with type II diabetes mellitus and hypertension.

Anthropometry

Weight: 58.4 kg

Height: 164 cm

Body mass index (BMI): 21.7 kg/m²

Medication

Actrapid slicing scale (four hourly)	Metronidazole
Dormicum	Clexane
Dobutrex	Aldactone
Diazepam	Lasix

Nutritional management and discussion

The patient was referred to the dietician for nutritional management.

A combination of nutritional guidelines was used, that is, guidelines for the management of critically ill patients (ESPEN¹ and ASPEN)² and those for the management of patients with diabetes mellitus (ADA).³

Biochemistry

	References values	16/08/2009	19/08/2009	03/09/2009
Sodium	135–147 mmol/L	145	146	140
Potassium	3.3–5.3 mmol/L	3.8	3.8	3.6
Chloride	99–113 mmol/L	117	112	99
CO ₂	23–29 mmol/L	21	26	34
Urea	2.6–7.0 mmol/L	15.8	4.9	4.6
Creatinine	60–100 mmol/L	131	66	50
Ca (Corr)	2.05–2.56 mmol/L	2.44	2.43	2.38
Mg	0.65–1.10 mmol/L	1.04	1.1	0.84
PO ₄	0.80–1.40 mmol/L	0.93	0.66	0.86
Albumin	35–52 g/L	29	18	12
Hb	12.1–16.3 g/dL	10.8	7.9	7.8
Hematocrit	0.37–0.49 L/L	0.32	0.23	0.24
WBC	4–10 10 ⁹ /L	18.7	14.6	8.8

It is recommended that energy requirements for ICU patients be calculated as follows:

- 20–25 kCal/kg/d during the acute and initial phases of critical illness (any energy excess should be avoided); and
- 25–30 kCal/kg/d during the post-acute phase and long-term stay.^{1,2}

The nutritional calculations were done using the actual body weight of 58.4 kg (since it constitutes a normal BMI).

The patient's nutritional requirements were calculated using an energy value of 22–25 kCal/kg/d, which is in line with the recommendations.

Non-protein energy (NPE): 1 343–1 460 kCal/d @ 22–25 kCal/kg

Protein: 70.1–87.6 g/d @ 1.2–1.5 g/kg/d

Carbohydrate: 182.5–219 g/d @ 50–60% NPE

Fat: 65–81.1 g/d @ 40–50% NPE

The goals of medical nutrition therapy for diabetes are as follows:

1. In the short term, attain and maintain optimal metabolic outcomes including the following:

- Maintain blood glucose levels in the normal range or as close to normal as is safely possible, avoiding either hypoglycaemia or hyperglycaemia and its associated risks of fluid imbalance, dehydration, ketoacidosis, hyperosmolar coma, infection and neurologic damage.
 - Maintain glycosylated A1c levels < 7%.
 - Maintain a lipid and lipoprotein profile that reduces the risk for macro vascular disease.
 - Maintain blood pressure levels that reduce the risk for vascular disease.
 - Aim for nitrogen balance.
 - Provide sufficient energy to meet the patient's needs and avoid the consequences of overfeeding.
 - Modify the hypermetabolic response, using pharmacologic doses of specific nutrients aimed at improving immune function, reducing inflammation and maintaining gut barrier function.
 - Maintain normal fluid and electrolyte balance.
2. In the longer term, prevent and treat the chronic complications of diabetes, for example dyslipidaemia, cardiovascular disease, hypertension and nephropathy.^{3,4}

Tetanus is a medical condition characterised by a prolonged contraction of skeletal muscle fibre, primarily caused by tetanospasmin, a neurotoxin produced by the gram-positive toxin-secreting clostridium *C. tetani*. Clinical features of the disease include general malaise followed by trismus due to masseter muscle spasm. The spasms may occur spontaneously but are easily precipitated by noise, light or handling of the patient. Respiration may be impaired because of laryngeal spasm; oesophageal and urethral spasm lead to dysphagia and urinary retention, respectively, and there is arching of the neck and back muscles. Autonomic dysfunction produces tachycardia, labile blood pressure, sweating and cardiac arrhythmias. Management of established tetanus includes supportive medical and nursing care. Patients are nursed in a quiet, isolated, well-ventilated, darkened room.⁵ The challenge of the medical nutrition therapy of this patient was to tailor the nutritional care plan to accommodate the energy expenditure from the spasm caused by tetanus as well as the manipulation of the macronutrient composition of the feed to reach the intervention objectives for type II diabetes mellitus and hypertension.

In keeping with current guidelines, feeding was initiated within 72 hours.^{1,2} Initially the patient received two litres of a special closed-system enteral feeding solution designed for diabetic patients. The feeding solution was administered continuously via a fine-bore nasogastric feeding tube. Certainly most 'standard' polymeric formulas, which contain approximately 50% carbohydrate, 30–35% fat and 15–20% protein as energy sources, could have been used. When faced with the nutritional management of critically ill patients, total energy intake may be more important than the macronutrient composition of enteral feeding solutions because of the metabolic abnormalities that are frequently seen in these patients.

However, a diabetic enteral feeding solution was given to this patient: The particular feed contained 33% monounsaturated fatty acids (MUFA) as well as EPA and DHA from fish oil. Diets containing up to 30% of total energy as MUFA have led to improvement in lipoprotein levels and glycaemic control in patients with both type I and type II diabetes mellitus.⁶ The feed also contained chromium, antioxidants and 2.4 g fibre/100 ml (66% soluble and 33% insoluble fibre). It is thought that some soluble fibre sources can slow glucose absorption from the small intestine, thus assisting with glycaemic control. One such study found that the addition of 50 g soluble fibre led to improved metabolic control when compared with 25 g fibre in obese subjects with type II diabetes mellitus.⁶ However, in the ICU setting, the tolerance of fibre sources is dependent on the function of the gastrointestinal tract, and use thereof (quantity and type) should be reconsidered based on the clinical setting. Patients at high risk of bowel dysmotility should not receive any fibre.² In the case being discussed, the resulting complications and the drop in albumin levels necessitated a change in the feeding strategy. A semi-elemental feeding solution was used at this stage to minimise gut effort and to enhance gut function.

Another important factor in the nutritional management of a diabetic patient is glycaemic control during enteral feeding since hyperglycaemia has been associated with an increased risk for infection in patients with diabetes mellitus and in ICU patients in general. Current guidelines for blood glucose control in critically ill patients with or without diabetes mellitus recommend keeping glucose values in the range of 6.1–8.3 mmol/L.^{2,4} There is a higher incidence of severe hypoglycaemia in patients whose blood glucose concentration is maintained between 4.5 and 6.1 mmol/L.²

References

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