Nutrition management of a trauma patient with pneumatosis intestinalis

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Background information

A 34-year-old man was admitted to the trauma unit after being involved in a motor vehicle accident, when the truck that he was driving overturned and entrapped him. Upon admission, the patient presented with blunt abdominal and chest trauma, a head injury and bilateral haemopneumothoraces. He was subsequently intubated and ventilated. The patient went into septic shock and required inotropic support.

The patient was taken to theatre for an exploratory laparotomy. A grade 5 splenic injury was discovered and a splenectomy was subsequently performed. The patient was readmitted to the intensive care unit (ICU) postoperatively. A nasogastric tube was inserted and put on free drainage. Clinically, the patient appeared to be well nourished on admission. His weight was estimated to be 85 kg and his height to be 185 cm (using the bed length). From this, his body mass index (BMI) was calculated to be 24.8 kg/m², reflecting the clinical impression. His serum lactate was significantly raised (6.2 mmol/l) on admission, and the urea, creatinine and liver function tests were mildly raised.

After two days of free drainage (300 ml), a semielemental feed was commenced via the nasogastric tube. It was infused continuously and the goal rate of 93 ml/hour was reached, providing 18% protein, 57% carbohydrate and 25% fat. The patient self-extubated in the early hours of the morning on day 4. He had to be reintubated, possibly as a result of aspiration. Consequently, his feeds were placed on hold and were restarted at midday.

On day 6, he started to show signs of feeding intolerance at goal rate, with vomiting and a distended abdomen. A rectal examination was ordered to exclude faecal compaction and a prokinetic agent (metoclopramide 10 mg three times a day) was administered intravenously. After being placed on free drainage, 3 300 ml of fluid was drained over the following 24 hours. Mesenteric ischaemia was suspected. A computed tomography (CT) scan and an ultrasound of the abdomen were performed on day 7. The results showed that the patient had pneumatosis intestinalis. He was kept on free drainage. Total parenteral nutrition (TPN) was subsequently started on day 8 (full rate providing protein 19%, carbohydrate 41% and fat 40%). By this time, the serum urea and creatinine were both raised (20.2 mmol/l and 150 µmol/l respectively). His serum lactate had normalised over the past few days, but increased again to 3.6 mmol/l on day 7. The patient remained on TPN until day 12. A progress CT of the abdomen was conducted on day 12, by which time, the serum urea and creatinine had normalised. A decision was made to commence with enteral feeding, in combination with TPN. A feed containing immunonutrients (glutamine and antioxidants) was administered (full rate of 20 ml/hour providing protein 68%, carbohydrate 30% and fat 3%). Over the next 2-3 days, TPN was weaned and the semi-elemental feed was increased slowly. Tolerance was monitored closely. The goal rate of 93 ml/hour was reached on day 18 providing 18% protein, 57% carbohydrate and 25% fat.

The patient's requirements were adjusted on day 23 to provide for more protein (1.3 g/kg). He remained on a semielemental feed. Sachets of glutamine, mixed with water, were administered via the nasogastric tube. By day 27, he breathed spontaneously via his tracheostomy and no longer required ventilation. This led to an increase in his calculated requirements. He had been tolerating his feeds well and the decision to change to a standard polymeric feed was made on day 29. This provided 15% protein, 55 % carbohydrate and 30% fat. By day 32, he was diagnosed with cerebral salt-wasting syndrome. He was polyuric and hyponatraemic (126 mmol/l). As a result, his feed was changed to a concentrated product that contained more sodium and less volume, providing 20% protein, 45% carbohydrate and 35% fat.

After spending 42 days in ICU, the patient's albumin improved from 24 g/l on admission, to 30 g/l. His sodium remained low at 123 mmol/l. Later that day, still on nasogastric feeds, he was discharged to a hospital closer to home where he would need to be closely monitored by both the dietitian and the medical team.

Literature review

Trauma injuries result in profound metabolical alterations that start at the time of injury and persist until wound healing and recovery are complete. Irrespective of whether the incident is sepsis, trauma or surgery related, once the systemic inflammatory response is set in motion, the physiological and metabolic changes that result are similar and may lead to shock and other negative outcomes. These responses are variable and in part relate to the patient's age, previous state of health and pre-existing diseases, the type of infection and the presence of multiple organ dysfunction syndrome.[1] Distinct metabolic derangements may be present in severely injured patients. These are characterised by increased substrate utilisation and protein catabolism. There is an abundance of evidence that supports the concept that nutritional support is advantageous and improves imperative clinical outcomes in the critically ill patient.[2]

Abdominal injuries may lead to pneumatosis intestinalis, which is a condition in which gas is found in the submucosa or subserosa of the bowel wall. It can either be found in a linear or cystic form.[3] Pneumatosis intestinalis is an uncommon condition which has recently received more clinical attention because of improved radiographic identification. Pneumatosis intestinalis has been shown to be present in 0.37% of patients who have abdominal CT scans. The available literature mainly comprises case reports and small case study series, with only a few large retrospective studies.[4] The pathogenesis of pneumatosis intestinalis is thought to be a result of many different contributory factors.

However, the development of pneumatosis intestinalis can be separated into two components:

- The mechanical aspect of gas transversing the mural portion of the bowel. This can be due to microbreaks in the mucosa, such as those caused by inflammation or necrosis. It can also be as a result of direct gas diffusion across an intact mucosal membrane, as can occur in situations of increased transabdominal pressure.
- Gas-forming bacilli entering the submucosa through mucosal tears or increased mucosal permeability, leading to the production of gas within the bowel wall, according to a bacterial theory.[4,5]

Initially distinguishing between life-threatening and benign causes of pneumatosis intestinalis is of extreme importance. Clinical symptoms and the laboratory records of patients are essential in determining its severity.[5] Mesenteric ischaemia is the most common life-threatening cause of pneumatosis intestinalis. There are also a number of traumatic or mechanical causes, one being blunt abdominal trauma. Benign causes of pneumatosis intestinalis can result from the side-effects of medication, most commonly from corticosteroids and steroids.[4,5] An elevated lactate level is a cause for serious concern about the development of ischaemia.[4] A recent study found that the combination of pneumatosis intestinalis and a serum lactic acid level > 2 mmol/l was associated with a > 80% mortality rate.[5]

Currently, there is no consensus on the appropriate clinical management of pneumatosis intestinalis. Algorithms have been created for its management. They are helpful, although tedious, and may be difficult to apply clinically when the patient requires a quick evaluation. As the aetiology of pneumatosis intestinalis varies greatly, management ranges from surgical intervention to outpatient observation.[4] Pneumatosis intestinalis is not a diagnosis in itself. Rather, it is the clinical presentation of an underlying disease.[3,5]

Nutritional management

For the initial calculation of the patient's requirements, the following were considered. For moderately to severely injured trauma patients (with or without TPN), energy requirements in the region of 25-30 kcal/kg body weight/day (105-126 kJ/kg/day)[2,6-8] should be used. Alternatively, Harris-Benedit-derived requirements multiplied by a stress factor of 1.3-1-5[2,6] can be used. O'Keefe et al recommend that protein needs to be estimated at at 1.5-2 g/kg/day.[2] The Society of Critical Care Medicine and American Society for Parenteral and Enteral Nutrition (ESPEN) recommend that in patients with a BMI < 30 kg/m^2, protein requirements should be in the range of 1.2-2 g/kg actual body weight per day, and may be even higher in multi-trauma patients.[9] The ESPEN guidelines state that when TPN is indicated, a balanced amino acid mixture should be infused at approximately 1.3-1.5 g protein or amino acid/kg IBW/day.[2,8] These recommendations were used as a guide for the calculation of the patient's energy and protein requirements. However, owing to renal insufficiency, a lower protein calculation of 1.2 g/kg was used initially.

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