Nutritional management of acute pancreatitis in a human immunodeficiency virus-infected patient

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Introduction

A 37-year-old male patient was referred from a local clinic with a one-day history of severe abdominal pain and difficulty breathing. In casualty, the patient reported pain in the right and left upper quadrant and the epigastrium, radiating to the back, accompanied by a retrosternal burning sensation. No nausea or vomiting was reported, but he had been constipated for the past three days. He was also known to have human immunodeficiency virus (HIV), and had been on highly active antiretroviral treatment (HAART) since 2008. On examination, the abdomen was distended, rigid and tender.

On admission, the patient's sodium and chloride levels were slightly decreased: 132 and 97 mmol/l, respectively. Albumin levels and haematocrit were towards the upper end of the normal range, indicating a mild degree of haemoconcentration. White cell count was elevated (12.13 x 10^9/l), while the serum amylase and liver enzymes were significantly elevated, i.e. amylase 1 643 units/l and alanine aminotransferase 110 U/l, aspartate aminotransferase 96 U/l, alkaline phosphatase 143 U/l and gamma-glutamyl transferase 206 U/l. The patient was diagnosed with acute pancreatitis, and sent to theatre for an exploratory laparotomy on day one. The surgical procedure showed haemorrhagic necrotic pancreatitis, a severely inflamed omentum, peripancreatic free fluid, as well as signs of fatty necrosis on the retroperitoneum. A nasogastric tube for free drainage was inserted, and the patient was kept *nil per os.* His oral antiretroviral drugs were also stopped. Postoperatively, he was awake and responsive, but in a critical condition with a poor prognosis. He was transferred to an isolation room in the general surgical ward as no beds were available in the intensive care unit (ICU). An antispasmodic [Buscopan® intravenously (IV) 10 mg three times daily], a histamine-2-receptor antagonist (Cimetidine IV® 200 mg twice daily), an analgesic (Omnopon® 20 mg three times daily), antibiotics (Rocephin® IV 1 g twice daily, Flagyl® IV 500 mg three times daily), and a motility agent (Maxalon® IV 10 mg three times daily) were prescribed. The patient's condition remained critical over the next three days and the nasogastric drainage remained well above 500 ml/day. The clinical course was characterised by persistent abdominal distension, temperature spikes, blood culture-confirmed bacterial infection and respiratory distress. Additional treatment included adapted antibiotics therapy (increased Rocephin® IV to 2 mg twice daily and Meronem® IV 1 g twice daily within eight hours), as well as oxygen therapy via a face mask.

The patient was referred to the dietitian on day three postoperatively once he had haemodynamically stabilised. A decision to start total parenteral nutrition (TPN) was made, and a central venous port (CVP)

Table I: Total parenteral nutrition recommendations and calculations pertaining to the patient

Energy or nutrient	Nutritional recommendation	Patient's calculated requirements	Nutrition provided by commercially available TPN and IV glutamine from day five postoperatively, onwards	Comment
Energy (kcal/day)	25-30 kcal/kg/day	1 875-2 250	1 980	Meets requirements
Nitrogen (g/day)	0.2-0.24 g/kg/day	15-18	19.2	Meets requirements
Glutamine (g/day)	0.3-0.5 g/kg/day	22.5-37.5	26.92	Meets requirements
Carbohydrate (g/day)	3-5 g/kg/day	225-375	250	Meets requirements
Fat (g/day)	0.8-1.5 g/kg/day	68-112.5	100	Meets requirements

IV: intravenously, TPN: total parenteral nutrition

requested. His albumin had decreased to 32 g/l after the laparotomy, but his urea and electrolytes profile was within the normal range. On day four, the patient was stable enough to be transported to the X-ray department for a confirmatory X-ray of the CVP location (mobile X-ray machines are only available in the ICU). TPN, as well as additional IV glutamine, were commenced on day four, and increased to the goal rate on day five. The patient's nutritional requirements and calculations for TPN are detailed in Table I.

Nasogastric drainage transiently decreased to 50 ml on day five, but increased dramatically to more than 1 000 ml/day on days six and seven while he was on continued TPN therapy. The patient complained of severe pain in the abdomen, and significant abdominal distension was noted. On day eight, the patient showed signs of wound dehiscence with herniation of a bowel loop which necessitated relaparotomy. During the latter procedure on day nine, a necrotic, gangrenous section of jejunum (approximately 30 cm) was removed and a primary end-to-end anastomosis performed. A nasogastric tube for free drainage was reinserted and initially less than 500 ml/day was drained on days nine and 10, but this increased to volumes of more than 1 000 ml/day on days 11-16. The patient's blood glucose also increased to above 14 mmol/l on day 11 and he was started on an insulin sliding scale. The patient passed flatus on day 15, and the abdominal distension improved. The nasogastric drainage started decreasing by day 17 and all antibiotics were stopped. The nasogastric drainage decreased to less than 200 ml/day on day 18. The nasogastric tube was removed and the patient started to mobilise. Semi-elemental feeds (30 ml every three hours taken orally) were ordered to test the tolerance of the enteral feeds on day 19, while the TPN was maintained at full rate.

The patient refused oral intake on day 20 and his condition started deteriorating. A rectal tube and a nasogastric tube for drainage were re-inserted to relieve the pressure in the abdomen. These drained 500 and 900 ml/day, respectively. A diagnosis of paralytic ileus was made, and the patient was started on an anticholinesterase (neostigmine 0.5 mg subcutaneously two times daily). The decision was taken to continue with TPN feedings only, and to stop all enteral feeds. This was continued for days 20-24, during which time the patient's condition improved slightly.

The patient developed severe diarrhoea on day 24 (six watery stools over the course of the day), which improved the following day (day 25 postoperatively). It was decided to once again test feed using oral semi-elemental feeds (30 ml every three hours) in addition to the TPN. Despite being counselled, the patient also consumed yoghurt and custard brought to him by his family. His diarrhoea was very severe on day 26 (five watery stools over the course of the day) and abdominal distension was noted. The patient also presented with elevated blood glucose values and temperature spikes. Enteral feeds were stopped, and TPN was continued at the full rate. The diarrhoea and abdominal distension had improved by day 31. The patient removed his CVP line and refused its reinsertion, threatening to sign refusal of hospital treatment. After he was counselled on his condition and prognosis, he agreed to stay in the hospital. As the patient refused IV lines, TPN could not be continued and oral semi-elemental feeds (30 ml every three hours) were started and were well tolerated. Oral polymeric sipfeeds (50 ml every three hours) were started on day 32 and were also well tolerated. The semi-elemental feeds were consequently stopped. The patient decided to eat soft porridge that his family had brought on day 33. This was well tolerated and a low-fat diet was ordered. In addition, the patient was restarted on HAART. The abdominal pain restarted on day 37, although no abdominal distension was noted. The patient was sent for a computed tomography (CT) scan of the abdomen, after which he was diagnosed with a pancreatic pseudocyst. Despite counselling by doctors in the ward, and against their advice, he signed refusal of hospital treatment on day 38 and discharged himself.

Literature review

In its healthy state, the pancreas is responsible for the secretion of enzymes that assist in the digestion of starch, lipids and protein, and which are secreted in response to food intake.[1] If these enzymes are activated prematurely, the pancreas becomes inflamed and necrotised as a consequence of autodigestion.[2,3] Inflammatory disease of the pancreas can be classified as either acute or chronic in nature (acute or chronic pancreatitis).[1] Patients experiencing acute pancreatitis usually complain of severe pain in the epigastrium, with occasional radiation to the back.[4] The patient has to meet at least two of the following criteria for a diagnosis of acute pancreatitis to be made: typical pain in the upper abdomen, serum levels or amylase or lipase exceeding three times the upper limit of normal, and CT imaging analysis confirming the diagnosis.[1,4] Discovering the aetiology of acute pancreatitis is critical for both the management and prevention of recurrent episodes.[1] While alcoholic and biliary causes account for the majority of acute pancreatitis cases, a number of other causative factors may be involved.[1] The acute inflammation in this patient may have specifically been the outcome of medicinal treatment (antiretroviral drugs like didanosine, acyclovir, lamivudine and stavudine) and/or infectious causes, including HIV.[1,2]

Patients with HIV are at a far greater risk of developing acute pancreatitis, which may relate to the following three factors:[2]

- The direct toxic effects of antiretroviral drugs on the pancreatic cells.
- Immunodeficiency, which predisposes such individuals to pancreatic infections, and worsens their prognosis. Therefore, the progression of HIV increases the risk.
- Alcohol abuse in the patient population affected by HIV.

Problems such as hypoalbuminaemia and anaemia are more common in HIV-infected patients, as are HIV-related symptoms like diarrhoea, fever and hepatomegaly. The hospital stay is often prolonged because of higher morbidity that relates to nosocomial infections, with a trend toward higher mortality. Complications like pancreatic pseudocysts, as well as respiratory and multi-organ failure may be seen. The discontinuation of the pancreatotoxins (in this case the HAART) is of extreme importance in drug-induced pancreatitis.[2]

Laboratory investigations that confirm the diagnosis of acute pancreatitis include elevated serum amylase and lipase concentrations, although only one of these tests is required for a diagnosis. Although a number of tests are available to determine the severity of acute pancreatitis, only haematocrit and C-reactive protein (CRP) are routinely available. A haematocrit over 44% on admission, and a CRP > 150 g/l, 48 hours after the onset of symptoms are indicators of necrotising pancreatitis. Unfortunately, no CRP was available for the patient in this timeframe, but his haematocrit was significantly above this level, indicating haemoconcentration and a poor prognosis.[1]

According to the 1992 International Symposium on Acute Pancreatitis that took place in Atlanta, acute pancreatitis can be classified as severe if the patient meets any one of the following criteria:[1]

- Organ failure, including at least one of the following: shock, pulmonary insufficiency, renal failure and gastrointestinal bleeding.
- Local complications, including necrosis, or an abscess or a pseudocyst.
- Systemic complications, including severe metabolic disturbances or disseminated intravascular coagulation.
- Three or more of the Ranson's criteria, or eight or more of the Acute Physiology and Chronic Health Evaluation II criteria.

This patient had severe pancreatitis since he suffered from pulmonary insufficiency and necrosis of the jejunum. Later, the patient also developed a local complication in the form of a pseudocyst. A pseudocyst consists of fluid, tissue, pancreatic enzymes, blood and debris, and usually develops 4-6 weeks after the onset of pancreatitis. It may resolve spontaneously or may have to be drained.[1]

The management of acute pancreatitis includes adequate fluid resuscitation, analgesia and prevention of organ failure. Pancreatic necrosis increases the risk of mortality, and surgery may be necessary, as was the case for this patient.[1]

Anthropometry

The patient reported weighing approximately 82 kg, three months prior to admission. His weight on referral was estimated to be approximately 75 kg. This indicates that he lost approximately 10% of his body weight in the preceding three months. His bed length was measured to be 1.85 m, leading to an assessment of a body mass index of 23.4 kg/m^2, which was in accordance with his physical appearance. Therefore, the patient's actual body weight was used in all calculations.

Nutritional management

Although the preferred route of nutritional support for patients with acute pancreatitis who require nutritional support is the enteral route, and not the parenteral route,[5] this patient's situation was complicated.

When the enteral route is chosen, peptide-based (semi-elemental) feeds, preferably containing medium-chain triglycerides, are recommended as polymeric feeds stimulate the pancreas and may result in severe pain.[5,6] The more recent consensus guidelines indicate that nasogastric feeds may be given,[5] although jejunal feeds may be better tolerated, particularly in patients suffering from severe acute pancreatitis that includes inflammation of the retroperitoneum.[6] Since the patient was in urgent need of nutritional support at the goal rate because of the pre-morbid significant weight loss, as well as the surgical intervention (laparotomy) which showed evidence of peripancreatic fluid collection, inflammation and necrosis, as well as the persistently high nasogastric drainage, it was decided to commence parenteral nutrition (PN) via a CVP. The peripheral route was not considered for this patient since he was uncooperative and frequently pulled out his peripheral IV lines. Although PN can be associated with an increased risk of severe hyperglycaemia, catheter-related sepsis and metabolic disturbances, if nutritional requirements are not controlled, it attenuates stimulation of the pancreas and the associated secretions, and improves the patient's nutritional status.[7,8]

The international consensus guidelines recommend that patients with severe pancreatitis should receive early nutrition therapy.[5] The American Society for Parenteral and Enteral Nutrition (ASPEN) guidelines for adult critically ill patients state that the first 24-72 hours following hospital admission, or the commencement of hypermetabolism, provide a window of opportunity for feeding.[9] The ESPEN guidelines on PN in pancreatitis also recommend that PN, if indicated, should be started as soon as possible, but only after adequate fluid resuscitation has taken place and once the patient is haemodynamically stable.[8] This was only achieved between 24-48 hours after the initial laparotomy. The pain and inflammation associated with pancreatitis result in an increase in the basal metabolic rate, which is linked to a higher energy requirement.[6,7] Hypermetabolism increases in relation to the severity and complications of a given clinical setting, and may be up to 40% higher than the predicted energy expenditure.[7] In this case, acute pancreatitis with sepsis were further contributors to an elevation in energy requirements, as well as the patient's chronic HIV infection and surgerical interventions.[10] The energy recommendations for PN in acute pancreatitis should be calculated using 25 non-protein kcal/kg/day, and should not exceed 30 kcal/kg/day.[5,8] This is in line with the guidelines for enteral nutrition for acute pancreatitis, as well as the enteral and parenteral nutrition guidelines for surgery.[6,11] It was decided to use the lower recommended range (25 non-protein kcal/kg/day) to decrease the risk of hyperglycaemia and increases in serum triglyceride levels in this patient. Overfeeding, which is not generally recommended, should be avoided in this particular clinical setting.[5,8]

In addition to hypermetabolism, patients experience an increased protein catabolism because of impaired protein synthesis and lower sensitivity to the protein-sparing effects of glucose.[7,8] An adequate nitrogen supply is imperative, and the goal for patients with severe acute pancreatitis is 0.2-0.24 g nitrogen/kg/day, or an equivalent

of 1.2-1.5 g amino acids/kg/day.[5,8] Glutamine supplementation is recommended at dosages of 0.3-0.6 g/kg alanyl-glutamine (Ala-Gln) dipeptide since it plays an important role in metabolic processes, resulting in a reduction of overall complications and a shorter hospital length of stay.[5,8] The patient requirements were met by the glutamine contained in the PN solution, as well as additional intravenous glutamine in the form of the Ala-Gln dipeptide.

The carbohydrate metabolism in patients with acute pancreatitis is altered as these patients may have increased insulin resistance which is associated with an increased risk of hyperglycaemia.[7,8] Nevertheless, glucose is the preferred energy supply,[5,8] and the maximal level of glucose oxidation is approximately 5-6 g/kg/day.[8] Preferably, the glucose should be administered at dosages between 3 g/kg/day and 5 g/kg/day, contributing between 50% and 70% of the total energy.[8] If necessary, exogenous insulin should be used to maintain blood glucose levels close to the normal range.[8] Glucose was calculated to provide approximately 50% of the total energy to prevent overfeeding and excessive hyperglycaemia in this patient.

Although glucose is the preferred energy supply, patients with acute pancreatitis are also more dependent on the products of fatty acid oxidation as energy substrates.[7,8] If the risk for triglyceridaemia is to be minimised, intravenous lipids should be given, considered to be safe for use in patients with pancreatitis.[5,8] Infusion rates should not exceed 1.5 g/kg/day.[8] The infusion rate for this patient was calculated as 1.3 g/kg/day. It is recommended that serum triglyceride levels are kept at < 4.6 mmol/l in patients on PN,[5] and < 12 mmol/l in patients with acute pancreatitis.[8]

The micronutrient recommendations for patients suffering from acute pancreatitis are no different to those for other critically ill patients. A daily dose of multivitamins and trace elements is recommended.[8] Although it has been suggested that tissue damage caused by free radicals contributes to the pathogenesis of acute pancreatitis, and thus, antioxidant supplementation may be beneficial,[10] insufficient evidence is available to recommend the supplementation of supranormal micronutrient dosages.[8] Therefore, the patient received vitamins and trace elements at the generally recommended supplementary levels as part of his daily TPN regimen.

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