Case Study: Intra-abdominal hypertension

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Introduction

A 34-year-old man with no significant past medical history was admitted to hospital on 16 February, with a three-day history of right lower quadrant pain, followed by nausea and vomiting. On physical examination, he appeared to be dehydrated (dry mucous membranes) with low-grade pyrexia [38˚C, tachycardia (102 beats per minute)], mild abdominal distension, reduced bowel sounds and maximal tenderness at McBurneys's point.

Abdominal radiography revealed a small bowel dilatation and air fluid levels, and was followed by abdominal sonography suggesting features of a perforated appendicitis. The gut appeared to be aperistaltic, and the appendix was non-compressible and dilated (outer diameter of 10 mm), with free fluid in the abdomen. The patient was subsequently taken to theatre for an exploratory laparotomy on 17 February. During the operation, the appendix was found to be perforated and gangrenous, with four-quadrant peritoneal contamination and distended loops of small bowel. A large faecolith was noted at the appendiceal base. An appendectomy was performed, with extensive peritoneal washout and a pencil drain placed. The abdominal fascia was closed, and the skin left open to heal by secondary intention. While closing the abdomen, raised intra-abdominal pressure was noted, and owing to concerns about the development of abdominal compartment syndrome, the patient was transferred to the intensive care unit (ICU).

Anthropometry

The patient's recumbent height was measured to be 165 cm, and his weight estimated to be 75 kg, with a body mass index (BMI) of 27.5kg/m^2. This correlated well with his clinical appearance. His ideal body weight was calculated to be 68 kg (based on a BMI of 25 kg/m^2).

Case report

On arrival in the ICU, the patient was intubated and placed on mechanical ventilation with fraction of inspired oxygen (FiO_2) of 0.4. He was haemodynamically stable and did not require inotropic or vasopressor support. A laboratory evaluation revealed an elevated C-reactive protein (CRP) of 287 mg/l and hypoalbuminaemia of 22 g/l. His blood glucose was well controlled at 6-8 mmol/l. On physical examination, the abdomen was distended and tender. Trans-bladder measurements revealed raised intra-abdominal pressure, ranging from 20-24 mmHg. He also presented with mild metabolic acidosis and a tapering urine output, which responded to fluids and furosemide.

Immediate measures were taken to reduce the intra-abdominal pressure, and included:

- Enteral decompression with a nasogastric tube.
- Initiation of prokinetic therapy to evacuate the intraluminal contents.
- Percutaneous catheter drainage to remove the intraperitoneal fluid collections.
- Diuretic therapy to remove excess fluid.

The patient was kept *nil per os* (NPO) and received intravenous (IV) fluid therapy in the form of lactated Ringer's solution. He was placed on a broad-spectrum antibiotic (amoxicillin plus clavulanate), antifungal (fluconazole), and received analgesic (morphine), antithrombotic (clexane) and ulcer prophylactic (ulsanic) therapy.

On day 2, the patient was taken back to theatre for a relook laparotomy since the laboratory analysis showed a dramatic rise in CRP (540 mg/l), together with persistently raised intra-abdominal pressure, ranging from 13-21 mmHg (normal range 5-7 mmHg in critically ill adults).[1] During the laparotomy, a diagnosis of intra-abdominal sepsis was made, and a pus specimen was sent for culture and sensitivity testing. The abdominal fascia was once again closed and the skin left open. Arriving back in ICU, the patient remained haemodynamically stable with a mean arterial pressure (MAP) of greater than 65 mmHg. Antibiotic therapy was escalated to tazocin (a combination of piperacillin and tazobactam), while awaiting the culture sensitivity results. The nasogastric tube was still on free drainage and had drained 300 ml over the past

24 hours. Furthermore, the patient presented with hypernatraemia (152 mmol/l). IV fluid therapy was changed to 5% dextrose water. At this point, the patient was referred to a dietitian for initiation of trickle feeding. An isotonic, fibre-free, semi-elemental feed was prescribed at a rate of 10 ml/hour. Prokinetic therapy in the form of erythromycin and metoclopramide was continued, and four-hourly gastric residual volumes measured. Culture sensitivity results confirmed the presence of *Escherichia coli,* bacteriodes and *Klebsiella* sensitive to carbapenems, a class of β-lactam antibiotics. Antibiotic therapy was subsequently escalated to ertapenem, a broad-spectrum carbapenem antibiotic.

Over the next two days (days 3-4), the patient remained septic, a diagnosis based on pyrexia (>38.3°C), tachycardia (>90/min^{-1}), tachypnoea (>20 beats per minute on continuous positive airway pressure (CPAP) ventilation), leucocytosis (white cell count >12 x 10^9/l), as well as an elevated creatinine (>44.2 µmol/l rise) and tapering urine output, the latter indicating severe sepsis with organ (renal) dysfunction.[2] Despite persistently high intra-abdominal pressure in the range of 18-26 mmHg, there was no marked abdominal distension.

Laboratory and blood gas analysis also showed a gradual decline in CRP, as well as an overall improvement in the acid-base status. A decision was made by the attending physician to continue nonoperative management and increase the feeding rate to 30 ml/hour. However, enteral nutrition (EN) was discontinued a few hours later because of an acute episode of vomiting and a measured gastric residual volume of 280 ml. The patient was kept NPO for the rest of the day. On day 4, since the patient was normotensive with a gastric residual volume < 200 ml, and the abdomen was non-tender and soft, the multidisciplinary team decided to restart trickle feeding (combined with the prokinetic therapy previously prescribed), but refrained from increasing the feeding rate owing to the risk of gut ischaemia associated with enteral feeding in the midst of persistently raised intra-abdominal pressure. By day 5, the intra-abdominal pressure remained high (range 18-22 mmHg), and supplemental parenteral nutrition (PN) therapy was initiated in order to achieve adequate nutrient intake (Table I). An electrolyte-free PN regimen was ordered because of the presence of hypernatraemia. Additional glutamine supplementation was not considered because of the presence of severe sepsis with renal dysfunction. Since the dietitian was not on call over the weekend, clear notes regarding the feeding plan were recorded on the patient's ICU chart. The plan was to increase the enteral feeding rate as soon as the intra-abdominal pressure remained below 20 mmHg, given that the patient was haemodynamically stable and showed no other signs of feeding intolerance, i.e. a persistently high gastric residual volume, abdominal distension or vomiting.

By Sunday (day 7), the measured intra-abdominal pressure was below 20 mmHg. Furthermore, the patient showed no signs of feeding intolerance and passed two soft stools. The EN formula was subsequently changed to a polymeric, isotonic, low-electrolyte formula, and titrated as per protocol to 70 ml/hour (25 kCal/kg, 1 g proteing/kg). PN was weaned accordingly, and stopped later on the same day.

Early Monday morning (day 8 at 02h00), the patient developed a sudden onset of sepsis-induced hypotension with subsequent tissue hypoperfusion. This led to a further decline in renal function (acute oligouria and metabolic acidosis), as well as acute respiratory distress syndrome (ARDS) (PaO_2/FiO_2 < 200). The patient's pulse rate was 143 beats per minute, respiratory rate 42 (on CPAP ventilation), and his initial blood pressure 95/42 with a MAP of 60 mmHg. EN was discontinued owing to haemodynamic instability (MAP < 65 mmHg). Goal-directed, protocol-driven resuscitation was initiated, which included inotropic and vasopressor support, and the administration of boluses of lactated Ringer's solution to increase the MAP to > 65 mmHg. By the time the dietitian assessed the patient (Monday at 08h00), he was awake and responsive, and maintained a MAP > 65 mmHg on a stable dose (0.11 µg/kg/minute) of adrenaline. However, he remained pyrexic (maximum temperature of 39.8°C, managed by active cooling) and because of several episodes of hyperglycaemia (> 10 mmol/l), was placed on a continuous insulin infusion to maintain his blood sugar level below 8 mmol/l. A laboratory evaluation showed a white cell count of 20.69 x 10^9/l, blood urea nitrogen of 28.3 mmol/l, creatinine of 273 µmol/l, hypocalcaemia (1.96 mmol/l), severe hypernatraemia (162 mmol/l) and hyperchloraemia (132 mmol/l). IV fluid therapy in the form of 5% dextrose water (60 ml/hour), with additional sodium bicarbonate, was administered to improve renal perfusion and treat metabolic acidosis and hypernatraemia. EN was restarted (the same EN formula as that given the previous day), and increased as per protocol to 70 ml/hour, providing 25 kCal/kg (29 kCal/kg, including 5% dextrose water), 1 g protein/kg, and 420 mg Na/day. Measured gastric residual volume was < 200 ml throughout.

By the next day (ICU day 9), the patient was anuric and required renal replacement therapy (RRT) in the form of sustained low-efficiency dialysis (SLED). IV fluid therapy was stopped, and fluid intake derived from enteral feeds alone. The patient was still on a continuous insulin infusion for blood glucose control. Furthermore, he remained hypernatraemic (155 mmol/l), pyrexic (maximum temperature

Table I: Feeding prescription (day 5)

Feeding prescription	Rate (ml/hour)	Total energy (kCal)	Non-protein energy (kCal)	Protein (g)	CHO (g)	Lipids (g)
Target (per kg/day)		25-30 (initially 25)	20-25 (initially 20)	1.2* (13 g N/day)	3-5	0.7-1.5
Trophic feed	10	240	198	11	34	7
PN regimen	60	1 144	891	63 (10.6 g N/day)	95	47
5% dextrose water	60	288	288	0	72	-
Total	120	1 672	1 377	74	201	54
Total (per kg/day)	45**	25	20	1.1	3	0.8

CHO: carbohydrates, N: nitrogen, PN: parenteral nutrition
*: 1.2 g protein/kg. Protein was restricted to 1.2 g/kg because of the presence of renal dysfunction or acute kidney injury, and no dialysis
**: 45 ml/kg. Intravenous fluid challenge, together with diuretics, to improve urine output (acute kidney injury)

Table II: Feeding prescription (day 9)

	Rate (ml/hour)	Total energy (kCal)	Protein (g)	CHO (g)	Lipids (g)	Sodium (mg)
Target (per kg/day)	- *	25 (up to 30**)	1.5 g/kg + 0.2 g/kg***	40-50% NPE	50-60% NPE	-
EN formula	45	1 620	108	133.92	72.36	513 (22 mmol)
Total (per kg/day)	-	24	1.6 g/kg	45% NPE	55% NPE	-
% of target	-	96%	94%	-	-	-

CHO: carbohydrates, NPE: non-protein energy; EN: enteral nutrition
*: Fluid target: No fixed fluid target. Determined by attending physician and aimed at avoiding fluid overload, as well as dehydration during dialysis, fever and sepsis
**: Fever further increases energy expenditure (10% increase for every 1°C > 37.5)
***: To compensate for increased losses via dialysate

of 39.5°C), and also developed severe diarrhoea of sudden onset. Prokinetic therapy was discontinued, and owing to concerns about possible *Clostridium difficile* infection, a stool sample was sent for *C. difficile* culture and *C. difficile* therapy (Flagyl®) was initiated empirically. A low-sodium and energy- and protein-dense feed was prescribed to allow adequate energy and protein provision at a fairly low rate of 45 ml/hour (Table II), given the presence of acute kidney injury (AKI) and fluid overload.

Table II details the patient's feeding prescription on day 9.

The next day (ICU day 10), the patient's septic condition was unchanged. A post-dialysis computed tomography (CT) scan of the abdomen ruled out the presence of intra-abdominal collections and nonoperative management was continued. Measured gastric residual volume remained < 200 ml, and there was a slight improvement in diarrhoea (*C. difficile* results pending). Furthermore, blood glucose control improved (< 8 mmol/l) with subsequent discontinuation of the continuous insulin infusion. The patient was kept on the same feeding prescription. In the early hours of the next morning, he required urgent RRT for sudden refractory hyperkalaemia (>7 mmol/l), but was considered to be too unstable for dialysis. The patient demised shortly thereafter, and post-mortem findings confirmed features of multi-organ failure with no intra-abdominal collections.

Discussion

Severe, life-threatening intra-abdominal infection, also known as peritonitis, is a frequent and dangerous entity in the ICU associated with increased morbidity, mortality and healthcare costs.[3] According to Eckmann et al,[4] one in four cases of severe sepsis or septic shock is caused by intra-abdominal infection. Almost 90% of all intra-abdominal infection is so-called secondary peritoneal infection, i.e. contamination of the peritoneal cavity, in this case following a perforated appendicitis, and requires a surgical approach primarily, i.e. appendectomy and peritoneal washout. Furthermore, a perforated appendicitis is also a risk factor for the development of intra-abdominal hypertension and abdominal compartment syndrome, in severe cases.[1]

Intra-abdominal pressure can be defined as the steady-state pressure within the abdominal cavity.[1] Although various methods are available for its direct and indirect measurement, the gold standard is via a urinary bladder catheter.[5] Although often measured in cmH_2O, the recently updated consensus definitions and clinical practice guidelines from the World Society of the Abdominal Compartment Syndrome Society (WSACS), state that intra-abdominal pressure should be expressed in mmHg (1 mmHg = 1.36 cmH_2O).[1] Intra-abdominal pressure is approximately 5-7 mmHg in critically ill adults. Intra-abdominal hypertension can be defined as a sustained or repeated pathological elevation in intra-abdominal pressure > 12 mmHg.[1]

Possible risk factors that may have contributed to elevated intra-abdominal pressure in this patient included:[1]

- Diminished abdominal wall compliance secondary to abdominal surgery.
- Increased intraluminal contents secondary to possible postoperative ileus.
- Increased intra-abdominal contents secondary to abdominal distension, intra-abdominal sepsis and/or intraperitoneal fluid collections.
- Capillary leak secondary to the severe inflammatory response syndrome.
- Others or miscellaneous, e.g. mechanical ventilation.

On day 2, post relook laparotomy, EN was initiated in the form of a trickle feed. Trickle feeding, also known as trophic feeding, can be defined as a small amount of EN, typically prescribed at a rate of 10-20 ml/hour, and administered primarily to maintain gut trophicity and mucosal barrier function, rather than serve as an energy supplement.[6] Trophic feeding was considered for the following reasons in this particular patient. Firstly, the WSACS recommends that EN should be minimised in patients with intra-abdominal hypertension, since an increase in intraluminal contents may further increase intra-abdominal pressure.[1] Secondly, intra-abdominal hypertension is often associated with gastrointestinal dysfunction. Raised intra-abdominal pressure causes a reduction in electrical and mechanical motor activity of the intestine and the inhibition of contractile responses, thereby leading to gut dysmotility.[7] Furthermore, the increased pressure within the abdominal cavity causes a reduction in splanchnic perfusion, with subsequent gut hypoperfusion.[7] In turn, this may lead to bowel ischaemia, intestinal oedema, intramucosal acidosis, capillary leak and translocation of gut bacteria with further worsening of sepsis.[5,7] The delivery of full EN, through increased mucosal oxygen requirements for nutrient absorption, may further increase the risk of the aforementioned complications.[8] On the other hand, the administration of low-dose EN or trophic feeding places a reduced metabolic demand on the gut, and may also induce splanchnic vasodilation and improve gut perfusion, thereby reducing the risk of gut ischaemia.[7,8] Moreover, trophic feeding has been shown to attenuate gut atrophy and improve host defences, thus limiting bacterial translocation.[9,10]

Table III: Nutrition-related problems and considerations

Nutrition-related problems	Considerations
Hypermetabolic and hypercatabolic state (severe sepsis, metabolic acidosis and fever)	Increased nutrient requirements
Severe hypernatraemia	Low electrolyte feed
Severe diarrhoea	Soluble fibre Partially hydrolysed/semi-elemental
Hyperglycaemia	Soluble fibre High fat to carbohydrate ratio
Fluid overload	Consider concentrated enteral nutrition formula to ensure adequate nutrient delivery despite fluid restriction.
AKI requiring RRT	Compensate for increased protein and micronutrient losses via dialysate
Acute respiratory distress syndrome	Consider enteral nutrition formula with a high fat to carbohydrate ratio and an anti-inflammatory profile

RRT: renal replacement therapy

When initiating trophic feeds in a patient at risk of gut hypoperfusion, e.g. intra-abdominal hypertension, the choice of feed plays an important role. This is because the composition of the enteral formula affects the degree of splanchnic vasodilation and metabolic demand, with hyperosmolar and fibre-rich enteral formula resulting in significantly more stress on the gut's absorptive capacity.[11] It has been proposed that hypertonic (> 700 mOsm) or fibre-rich enteral formula draws fluid into the gut, predisposing the patient to diminished gut perfusion, as well as dysmotility and subsequent small bowel overgrowth.[6,8] Therefore, enteral formula with modest osmolality and minimal fibre may reduce the risk of complications relating to the feeding formula.[8] According to the American Society for Parenteral and Enteral Nutrition, soluble and insoluble fibre should be avoided in patients at high risk of bowel ischaemia or severe dysmotility.[12] Some studies even suggest that peptide-based formula may improve feeding tolerance through easier absorption across the gut lumen. Such formula may also improve nitrogen retention through increased visceral protein synthesis, as well as induce a trophic effect on the intestinal mucosa through increased glucagon synthesis.[13] For the aforementioned reasons, trophic feeding was administered in the form of an isotonic, fibre-free, semi-elemental formula in this patient.

On day 3, EN was increased to 30 ml/hour, but discontinued a few hours later owing to an acute episode of vomiting and a measured gastric residual volume of 280 ml. Currently, there is insufficient scientific evidence or physiological grounds to establish a clear-cut threshold for a high gastric residual volume. According to Verburgh et al,[14] gastric residual volume beyond 200 ml requires close monitoring. However, routine cessation of EN, solely on the basis of a gastric residual volume of 200-500 ml, should be avoided. EN was discontinued in this patient on the basis of a gastric residual volume > 200 ml despite prokinetic therapy, combined with an acute episode of vomiting and raised intra-abdominal pressure. Verburgh et al further recommend frequent challenges with small amounts of EN (evidence grade 2D) in patients with raised intra-abdominal pressure and/or consistent feeding intolerance, despite deliberate interventions taken to improve nutrient delivery, e.g. prokinetic therapy.[14] For this reason, trophic feeding was restarted the next morning (postoperative day 4). Based on the patient's pre-morbid nutritional status (BMI 27.5 kg/m^2), supplemental PN was only initiated on postoperative day 5. Supplemental PN is widely recommended as a valuable tool for the prevention of a growing protein energy debt in the case of persistent EN intolerance.[15] Nonetheless, the optimal timing of supplemental PN remains controversial, especially after the publication of recent trials,[16-20] with conflicting results on the respective merits of early versus late initiation of supplemental PN. Although further research is needed before formal recommendations can be established, optimal timing seems to be somewhere between days 3 and 7, taking into consideration the severity of the illness, expected ICU length of stay and nutritional status.[10,21-24] However, with combination feeding, daily monitoring is essential to avoid the potential risk of overfeeding.[9] Furthermore, non-nutritional energy sources should be included in the total energy count to avoid overfeeding. For example, hypernatraemia and/or metabolic acidosis are often treated with the administration of 5% dextrose water (free water), and depending on the prescribed rate, can add considerably to total energy intake.[25,26] Failure to do so may inevitably result in overfeeding, with further worsening of hyperglycaemia, as well as other potentially adverse effects, such as hypercapnoea, failure to wean from mechanical ventilation, azotaemia, immunosuppression, hypertriglyceridaemia and hepatic steatosis.[10,15,27-29]

Supplemental glutamine was not considered in this patient since glutamine should be avoided in ICU patients with multi-organ failure or shock, and should only be given to non-multi-organ failure patients receiving PN, based on the recently published REducing Deaths due to OXidative Stress (REDOXS) study by Heyland et al.[10,30-32]

From postoperative day 8 onwards, the patient required continuous inotropic and vasopressor support to maintain haemodynamic stability. Based on current evidence, vasopressors are not a contraindication to EN. Some studies even suggest that EN might benefit this type of patient subgroup through its ability to safely restore splanchnic perfusion and oxygenation.[6,8] According to Heyland and Dhaliwal,[33] the results of a large observational study of 1 174 critically ill patients on vasopressors revealed that early EN was associated with reduced hospital mortality, and that the beneficial effect of early EN was more pronounced in patients on

multiple vasopressor therapy. Therefore, the general consensus remains that EN can be safely administered in patients on stable or declining doses of vasopressors.[6,12]

On postoperative day 9, the patient's condition further deteriorated, and RRT in the form of SLED was initiated. Upon review of the patient's feeding prescription, various factors were taken into consideration (Table III).

Severe hypernatraemia precluded the dietitian from prescribing a semi-elemental feed because of its high sodium content, which would otherwise be considered in a patient with concomitant hypoalbuminaemia and diarrhoea. The only option available was a low-sodium and energy- and protein-dense feed. This allowed the provision of adequate energy and protein at a fairly low rate of 45 ml/hour, given the presence of AKI and fluid overload. Adequate nutrient provision is especially important in patients undergoing RRT, since together with the loss of vitamins and trace elements, an extensive amount of amino acids and protein may be lost through the extracorporeal circulation of RRT. On average, protein and amino acid losses, with the use of high-flux filtres and/or highly efficient modalities, such as SLED and continuous RRT, may be quantified as 0.2 g/kg of amino acid/litre of ultrafiltrate, amounting to 10-15 g amino acid/day, plus an additional 5-10 g/day of protein.[34,35] The high fat to carbohydrate ratio of the feed may improve blood glucose control, and along with its anti-inflammatory profile (omega-3 fatty acids; eicosapentaenoic acid and docosahexaenoic acid), may play a supportive role in the management of ARDS. Despite the high fat content of this particular feed, the 100% soluble fibre may have assisted in binding the stools (grade C)[12,14] and improving blood glucose control.[36] Tap water can also be administered as an hourly flush, in addition to the enteral feed, to assist in the management of hypernatraemia, and possibly to provide relief from the diarrhoea through the dilution of the concentrated enteral formula.

Conclusion

This case presentation reflects the dynamicity of critical illness and the importance of daily monitoring to ensure appropriate adjustment of feeding goals to meet constantly changing metabolic demands. Furthermore, it also highlights several controversial issues with regard to nutrient delivery in the ICU, i.e. the role of trophic feeding, optimal timing with regard to the initiation of supplemental PN, as well as glutamine administration in the critical care setting.

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