HORSES AND OTHER EQUIDS

Successful treatment of prolonged postoperative ileus following resection of a small intestinal spindle cell sarcoma in a horse

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SUMMARY

This case report details the unusual clinical course of a horse that had been hospitalised for recurrent colic prior to an acute colic episode requiring emergency laparotomy. Within the first 24 hours after surgery, the horse developed severe colic and postoperative ileus (POI) requiring repeat laparotomy. The horse suffered persistent ileus with copious reflux for 8 days thereafter, but its clinical condition permitted continued treatment. It was discharged after 4 weeks and returned to its previous performance level. The authors describe clinical decision-making regarding intravenous fluid therapy, prokinetic drug administration, enteral and parenteral nutrition, and institution of physical exercise in the treatment of persistent POI. Economic and welfare considerations allowing, equine POI may resolve even in protracted cases. Patients may benefit from strategies aimed at early enteral feeding and ambulation as have emerged in human POI management.

BACKGROUND

Postoperative ileus (POI), also known as adynamic or paralytic ileus, is a (temporary) disturbance in gastric and intestinal propulsive motility following surgery. It is a common complication after colic surgery in horses, with a reported incidence of 10%–50%[1] and high associated mortality. Strategies to reduce the incidence of POI in horses rely largely on medical therapy with parenteral fluids, prokinetic, analgesic and anti-inflammatory agents, as well as antibiotic prophylaxis.[2 3]

Based on human studies, management strategies aimed at early ambulation and re-introduction of enteral nutrition could prove equally important to help re-establish normal gut motility patterns and restore enterocyte nutrient and energy supply in patients with POI.[4 5] Although human guidelines stipulate avoidance of routine nasogastric intubation, horses' inability to vomit necessitates regular stomach decompression to avoid traumatic rupture.[6] Early resumption of enteral feeding likewise is precarious as this could aggravate gastric overload. Although hand-walking and timing of feeding were considered 'quite important' factors by 50%–86% of equine clinicians in recent surveys of POI management,[2 3] no controlled trials have addressed the efficacy of ambulation or re-feeding protocols for horses after colic surgery.

Prognosis for horses with persistent POI depends largely on the individual patient (clinical condition, cooperativeness, welfare considerations), the owner (motivation, financial means), and on the available facilities and staff required for long-term intensive care treatment. We report an unusual case of a horse presenting with severe acute colic and small intestinal (SI) strangulation, which developed POI requiring repeat laparotomy with reflux persisting for eight more days, but which was successfully treated and discharged 4 weeks after surgery. The clinical course with resolution of POI shortly after an increase in oral food intake and institution of treadmill exercise is described, and the evidence base behind the employed treatment strategies for equine POI is discussed.

CASE PRESENTATION

A 12-year-old, 700-kg Dutch warmblood Small Tour dressage horse was admitted to the Utrecht University Equine Internal Medicine Department because of intermittent colic over the preceding 3 months. During hospitalisation, a tentative diagnosis of colonic dysmotility was reached. The horse was treated with a diet change and paraffin and was discharged after 9 days. The day after discharge, the horse was returned to the Utrecht University Equine Internal Medicine emergency service with severe acute colic.

INVESTIGATIONS

On clinical examination, the horse was mildly depressed with increasingly frequent episodes of acute abdominal pain (pawing, kicking at abdomen, attempting to lie down). It had a pulse rate of 48/min, rectal temperature of 37.8°C, normal jugular refill time, pink mucous membranes with capillary refill time of 2 s and hypofrequent intestinal sounds. Laboratory results revealed a packed cell volume (PCV) of 36 L/L and lactate of 1.1 mmol/L with normal electrolyte and acid–base status. Based on clinical and laboratory findings, dehydration was estimated at 4% of body weight. Rectal examination revealed oedema of the cranial mesenteric root; palpation was consistently painful. Abdominal ultrasound showed several mildly distended, hypomotile SI loops. Abdominocentesis was not performed, as the decision for exploratory laparotomy was made based on the severity of colic signs that were refractory to detomidine (0.1 mg/kg intravenously) and butorphanol (0.05 mg/kg), combined with recent clinical history.

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DIFFERENTIAL DIAGNOSIS

Based on clinical presentation, rectal examination and abdominal ultrasound, a strangulating SI lesion was suspected (mesenteric hernia, mesenteric rent, volvulus intestinalis, pedunculated lipoma, intussusception (unlikely given age)).

TREATMENT

The horse received butorphanol (0.1 mg/kg; Dolorex, Intervet, the Netherlands) and detomidine (0.015 mg/kg; Domosedan, Orion, Finland) intravenously, as well as 6 L of Ringer's (Fresenius Kabi, Zeist, Germany), 6.5 mg/kg gentamicin (Eurovet, the Netherlands) and 2×10^6 IU benzylpenicillin–sodium (Eurovet). Anaesthesia was induced intravenously with diazepam (0.05 mg/kg; Centrafarm, the Netherlands) and ketamine (2.2 mg/kg; Narketan, Vetoquinol, France), after which maintenance was with isoflurane (Isoflo; Abbott Laboratories, UK) in oxygen, supplemented with constant rate infusions (CRIs) of ketamine (0.5 mg/kg/h) and lidocaine (Utrecht University Veterinary Apothecary; 3 mg/kg/h, loading dose 2.5 mg/kg intravenously) that were ceased 30 min before the end of surgery. During anaesthetic maintenance, 12 L Ringer's (Fresenius Kabi) was infused intravenously and dobutamine (Hameln pharma, Germany) CRI was titrated to effect (0.001–0.003 mg/kg/min) to maintain mean arterial pressure above 70 mm Hg. Surgery revealed an anti-mesenteric SI mass, located approximately mid-jejunum, with SI strangulation due to true mesenteric (not segmental) volvulus and marked bowel distension proximal to this site. The lesion itself only minimally reduced the luminal diameter. The fist-sized reddish-purple mass appeared lobular in structure, with cystic as well as more solid regions; it was resected with 20 cm of adjacent jejunum and submitted for histology. An end-to-end anastomosis was created and closed in a two-layer (continuous, inverting) suture pattern using Monocryl 2-0. Anaesthesia duration was 185 min and the horse received 0.002 mg/kg detomidine intravenously for recovery. After an uneventful head and tail rope–assisted recovery, the horse was admitted to the intensive care ward. An overview of the time course of the first 9 days of intensive care treatment is provided in figure 1.

Initial medical treatment consisted of CRI Ringer's (2 L/h) with electrolytes (KCl, Fresenius Kabi; Ca–Mg solution, Dechra, Bladel, the Netherlands) supplemented based on plasma chemistry results, lidocaine CRI (Utrecht University Veterinary Pharmacy, the Netherlands; 3 mg/kg/h), flunixin (Cronyxin, Crossvet pharma, Ireland; 1 mg/kg intravenously q12h), gentamicin (6.5 mg/kg intravenously q24h), procain penicillin (Procapen, AST Farma, the Netherlands; 20 mg/kg IM q24h) and dalteparin (Fragmin, Pfizer, the Netherlands; 50 IU/kg SC q24h). The first day after surgery, the horse appeared depressed with no appetite, and it developed enterogastric reflux (10–19 L q4h). Fluid rate was increased to 4 L/h (figure 2) and a CRI of metoclopramide

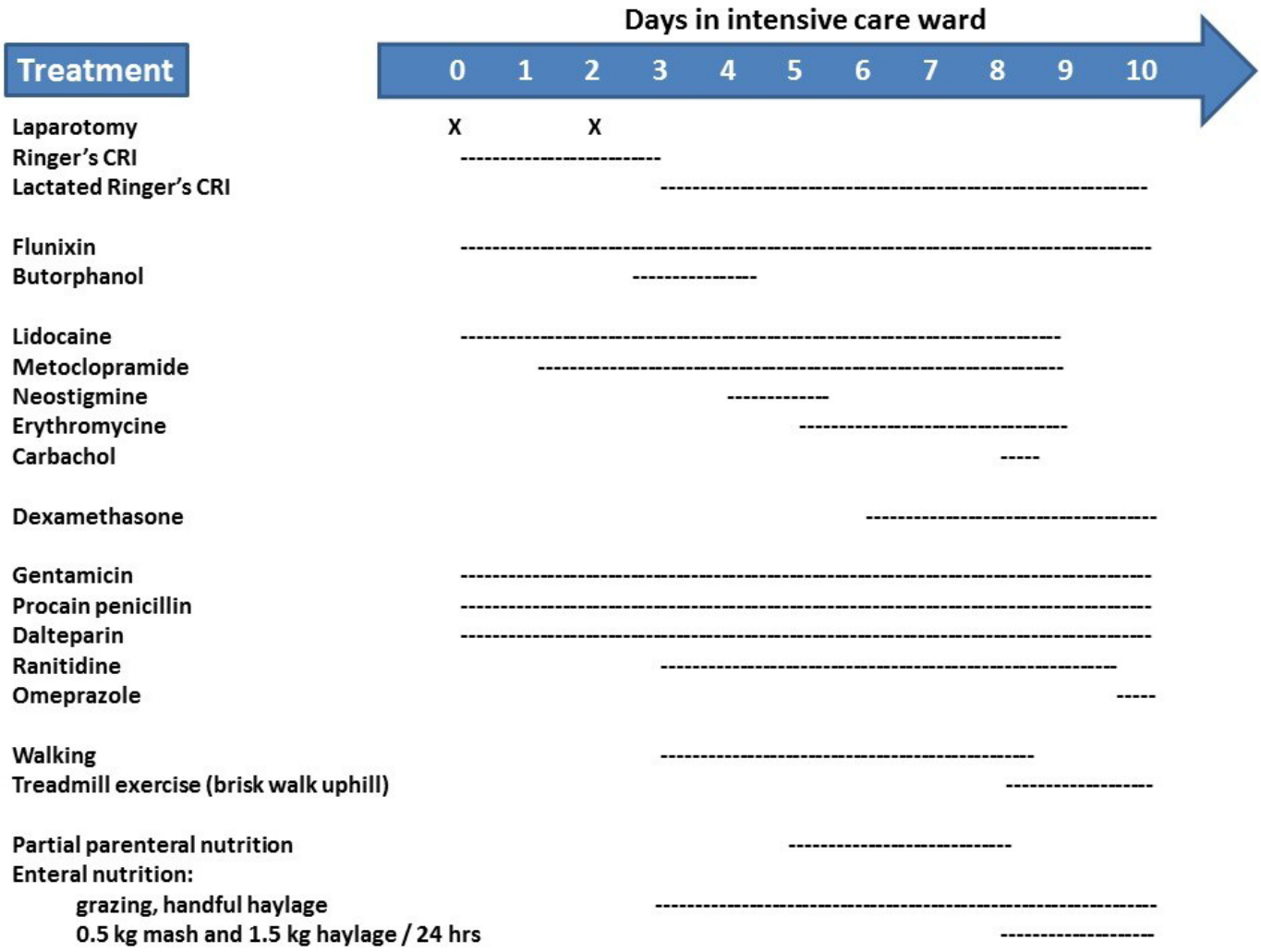

Figure 1 Overview of medical treatment and patient management during the first 10 days of intensive care ward hospitalisation.

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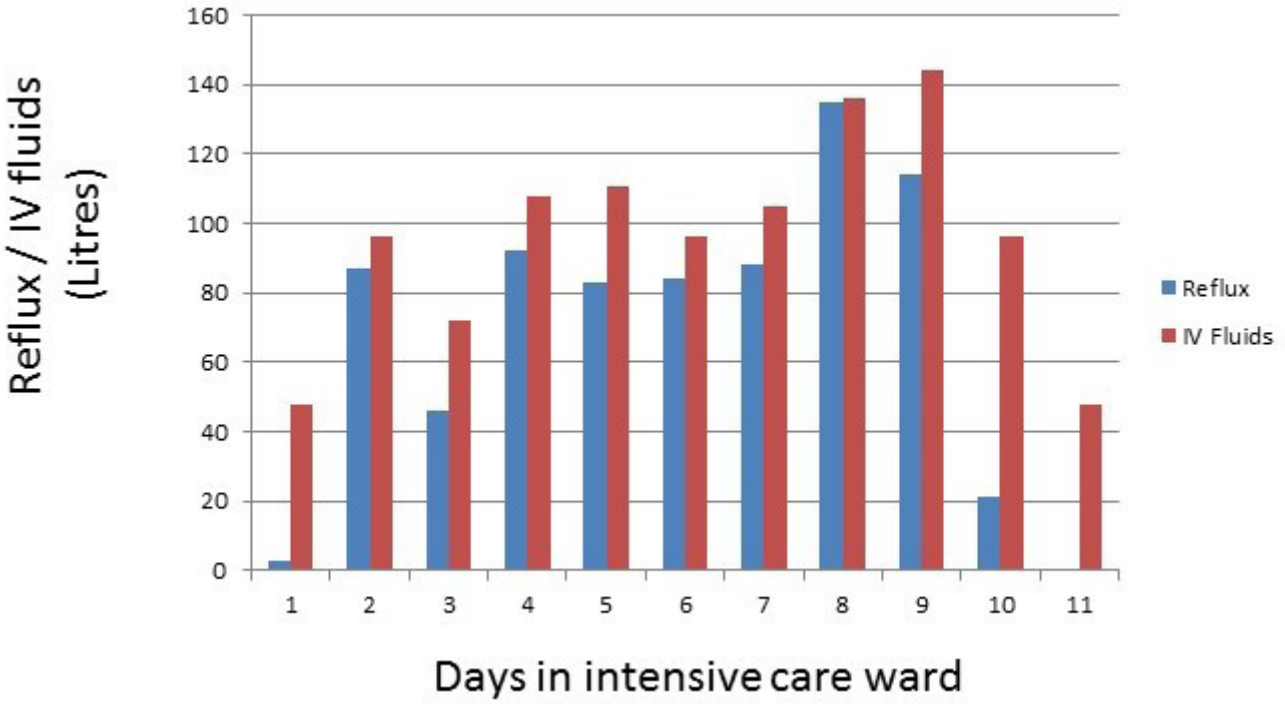

Figure 2 Volume of crystalloids infused and spontaneous reflux obtained via nasogastric intubation per 24 hours during the first 10 days of intensive care ward hospitalisation. Fluid therapy consisted of Ringer's solution on days 1 and 2 (supplemented with sodium bicarbonate, potassium chloride and calcium–magnesium gluconate infusions as needed based on clinical chemistry results), with Ringer's replaced by lactated Ringer's solution from day 3 onwards.

(Vomend; Eurovet) was started at 0.04 mg/kg/h. That night, the horse became increasingly restless with a pulse rate of 64/min. Pain score (EQUUS COMPASS[7]) was consistently above 10, and 20-L reflux accumulated in 4 hours. Abdominal ultrasound revealed severely distended SI loops (8 cm diameter, wall thickness 3 mm), PCV was 39 L/L and lactate was 1.6 mmol/L. The horse received detomidine 0.01 mg/kg and butorphanol 0.1 mg/kg intravenously with 9 L Ringer's as fluid bolus in preparation for repeat laparotomy for refractory colic associated with ileus that same morning. The second surgery revealed a patent jejunal anastomosis, acceptable SI motility and only a modest amount of fibrin. The ascending colon was distended with semisolid contents; 3 L water and 2 L paraffin (Eurovet) were administered into the caecum via a small enterotomy. The SI was decompressed into the caecum and the abdomen was closed. Recovery was uneventful and the horse returned to the ward on CRI lactated Ringer's (2–4 L/h), supplemented with lidocaine (3 mg/kg/h) and metoclopramide (0.04 mg/kg/h), with all other medications continued.

Within 12 hours, reflux returned and the horse kept refluxing quantities upwards of 12 L per 4 hours thereafter (figure 2). Signs of abdominal discomfort at this time were mild and responsive to butorphanol CRI (0.013 mg/kg/h). The horse's clinical condition permitted continued treatment, now supplemented with ranitidine (Zantac; GlaxoSmithKline, the Netherlands) 1.2 mg/kg intravenously q6h as a gastroprotectant. From day 3, the horse was hand-walked and allowed to graze for 10 min, multiple times daily, and small handfuls of haylage were fed just after stomach decompression. Butorphanol CRI was ceased and neostigmine (Centrafarm, the Netherlands) was started on day 4 (5 mg/kg SC q2h for four times) to treat mild colic caused by an ascending colon impaction, diagnosed by rectal palpation. Resolution of the impaction was confirmed by rectal examination on day 5, but distended SI loops were still palpable and reflux amounted to 14–22 L every 4 hours (figure 2). In close consultation with the owners, treatment continued and the horse was started on erythromycin (Erythrocin, Amdipharm, Ireland; 0.5, 1 and then 2 mg/kg intravenously in 3 L saline q8h) while Ringer's CRI was reduced to 3 L/h with fluid boluses only as needed, based on clinical signs and laboratory results. As the horse had lost 96 kg of bodyweight in 5 days, partial parenteral nutrition was started (Nutriflex; Braun, Germany); 100 mL/h for the first 8 hours, increasing to 200 mL/h over the first 24 hours and to 400 mL/h thereafter (DE calculated based on Stratton-Phelps,[8] with metabolic weight of $600^{0.75}=123$). This was administered via a dedicated 14G 9-cm over-the-needle catheter (Mila International, Erlanger, Kentucky, USA) in the right lateral thoracic vein.

The next day, SI distension was less (5–6 cm) and some motility was noted. However, severe SI wall thickening (up to 5–6 mm) was seen; dexamethasone (Rapidexon; Eurovet) was started at 0.06 mg/kg intravenously q24h. On day 7, reflux persisted but abdominal ultrasound revealed further improvement in SI contractility with unchanged SI wall thickness. The right lateral thoracic vein felt hardened and a new 14G catheter was placed in the left lateral thoracic vein for continued parenteral nutrition. That evening and the next morning (day 8), the horse's condition deteriorated; body weight had stabilised (11-kg weight loss in 3 days), but reflux increased to 22 L in 3 hours, mucous membranes were red and PCV was now 45 L/L despite continued fluid therapy. The left lateral thoracic vein was swollen; the catheter was removed and parenteral nutrition suspended. With owner informed consent, carbachol (Miostat; Alcon, the Netherlands) was started at 0.001 mg/kg SC q4h for three consecutive administrations. Also, it was decided to feed the horse a 1.5-kg portion of haylage, after which it was put on a treadmill on a 10% incline at a brisk walking pace. Immediately after 15 min of treadmill exercise, the horse defaecated for the

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first time in 8 days. Treadmill exercise was repeated at 6-hour intervals thereafter.

Reflux subsided the next day and subsequent night from 20 L/4 hours to 12, 8 and then 2 L, after which stomach decompression was discontinued. Enteral nutrition was maintained and increased gradually per day with mash (0.5 kg) but mainly haylage (1.5 kg/24 hours in handfuls). The next 3 days, the horse improved further but then suffered a severe clinical setback with fever and even a brief return of reflux, associated with a developing coliform incisional site infection. The third week, the horse developed a painful swelling in its right hindlimb, which ultrasound showed to be a venous thrombosis of the right lateral plantar vein; this was successfully treated with dalteparin (50 IU/kg SC q24h).

OUTCOME AND FOLLOW-UP

The horse was discharged after 26 days on a balanced daily diet consisting of 6 kg haylage, 4 kg low-carbohydrate feed (Fiber force; Cavalor, Belgium), 1 kg mash (Pavo slobber; Pavo, the Netherlands) with a protein, vitamin, mineral and fatty acid supplement (Pavo Vital Complete; Pavo) and continued dalteparin treatment (50 IU/kg SC q24h) for 2 weeks. The horse returned regularly for abdominal wound check-ups the first half year after discharge and despite multiple repeat colic episodes (all of which could be managed medically) successfully returned to his previous level of dressage training and progressed to grand prix competition, before being retired due to chronic distal limb lameness (follow-up period: 46 months).

DISCUSSION

Adynamic ileus after gastrointestinal (GI) surgery is believed to result from a disturbance in normal hormonal, vascular and neuromuscular control of GI motility, leading to disruption of propulsive activity.[9 10] SI wall inflammation, secondary to severe distension, ischaemia–reperfusion injury and/or surgical manipulation, is believed to be central to enteric nervous system and intestinal smooth muscle dysfunction in equine postoperative ileus.[6 11]

The patient of this report presented with SI strangulation at the site of an intramural mass, which proved to be a spindle cell sarcoma. Microscopically, the mass was classified as a low-grade sarcoma. Although locally invasive, it was deemed benign based on cellular differentiation. Importantly, the surgical margins were free of tumour cells. Intestinal neoplasia is an uncommon finding in horses. Three previous reports of intestinal spindle cell–type sarcomas causing recurrent colic and intestinal obstruction in five horses were identified in the literature.[12–14] On the basis of histopathology and the macroscopic appearance of the GI tract as examined during both surgeries, it was deemed unlikely that diffuse GI metastases were the primary cause of prolonged POI in this case.

Several factors likely contributed to the favourable outcome in this horse, not in the least the fact that the horse remained reasonably bright and cooperative during all treatments and the owners were highly motivated and expressed no financial limitations. Economic and welfare considerations are major reasons for owners to request euthanasia of horses with prolonged POI.[1] Repeat laparotomy was indicated given the return of SI distension and severe colic that was unresponsive to analgesics and sedatives within 24 hours after initial surgery. Although repeat laparotomy carries an 18-fold increased risk of postoperative mortality,[15] it is an accepted treatment option for POI cases that do not respond to medical therapy.[1] Neither mechanical obstruction nor severe peritonitis were identified during repeat laparotomy; enterogastric reflux returned unabated within 12 hours after recovery. Importantly though, colic did not return, and lidocaine CRI with twice-daily flunixin was sufficient for postoperative analgesia except on day 3, when butorphanol CRI was administered for dullness and mild inappetence. This was possibly secondary to suspected gastric ulceration (as multiple risk factors including performance horse, prolonged fasting, pain and stress were present) and/or developing colonic impaction, the latter of which was diagnosed by rectal palpation on day 4.

The standard approach adopted by our institution to POI treatment in colic horses in the absence of identified underlying systemic conditions (sepsis, (septic) peritonitis) consists of intravenous crystalloids with guided electrolyte correction to replace deficits and then match losses, lidocaine CRI, dalteparin and twice-daily flunixin. This is also the most commonly adopted strategy reported in the most recent survey of European and American specialist equine clinicians.[2 3] Although prokinetic drugs may seem a logical choice for the treatment of equine POI, there are only limited data and ongoing debate regarding their efficacy in clinical cases. Lidocaine CRI was started intraoperatively during the first laparotomy and continued thereafter. Our rationale for its use include its analgesic and anti-inflammatory effects[16] as well as the— disputed—potential for it to reduce the incidence, volume and duration of reflux in horses after colic surgery.[17 18] Metoclopramide was later added in an attempt to improve gastroduodenal coordination,[19] while neostigmine was started on day 4 to treat colonic impaction and suspected disturbed large intestinal motility.[20] Importantly, it is often unknown whether beneficial or detrimental effects seen with these prokinetic agents in healthy horses also occur in horses with pre-existing GI pathology and/or POI. Indeed, differential effects were reported in healthy horses compared with horses with GI disease when treated with intravenous erythromycin.[21] Administration of the latter may also be debated because of concerns regarding judicious antibiotic use and, like carbachol, it is not normally used as a prokinetic drug in our clinic. Since none of the prokinetic agents were used as stand-alone treatment, it is impossible to know in hindsight how much any of these contributed to the outcome in this case. Importantly, adverse effects like muscle tremors, excitation and abdominal pain, as well as drug interactions, have been associated with these agents, warranting judicious use, particularly when several agents are combined. No suspected adverse effects were noted in the patient of this report.

While attempting to stimulate GI contractility with cholinergic, anti-adrenergic and/or anti-dopaminergic agents may seem theoretically sound, these agents do not address the underlying cause of the prolonged disturbance in GI motility patterns. Inflammation within the *lamina muscularis* has been identified as a key factor.[9] Increased SI wall thickening was noted during abdominal ultrasound examination and was especially marked on day 6. This may have been caused by ongoing mural inflammation (primary and/or secondary to bowel manipulation during two consecutive laparotomies), but may also have been due to altered GI microvascular balance and/or patient overhydration.[9 22] To address the possibility for mural oedema being secondary to potential infiltrative enteritis in this patient, dexamethasone was administered intravenously at this time. As for the role of intravenous fluid therapy in intestinal oedema, several human guidelines currently recommend restrictive fluid regimes for GI surgery, aiming for near-zero fluid balance[23]; in fact, restrictive fluid therapy has also been linked to a reduction in incisional site

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infections after abdominal surgery in humans.[24] It can be very challenging to maintain near-zero fluid balance in large horses with unstable haemodynamics, losing copious amounts of fluid as reflux, and it is indeed possible that mural oedema seen at this stage in our patient was caused or aggravated by 'unmatched' or overzealous crystalloid fluid administration. The need for fluid therapy was estimated every 6–12 hours, based on ongoing losses and clinical cardiovascular parameters as well as results of haematology (PCV) and clinical chemistry (total protein, lactate, pH, base excess, electrolytes). Hypoproteinemia did not occur at any time, and fluid rate had already been reduced in an attempt to only match losses. Arguably, colloids or even hypertonic saline boluses could have been considered for this patient to help reduce the volume of fluids administered intravenously and perhaps reduce third-space loss,[9] but no evidence regarding the clinical efficacy of such an approach (or fluid rate to be administered) for limiting intestinal oedema in horses with POI is available to date.

An ultimate attempt to promote passage of solid contents to the duodenum and to restore the duodenal-gastric reflex was undertaken on day 8, with the patient fed 1.5 kg roughage just after stomach decompression and before being placed on a treadmill and made to walk briskly uphill. Remarkably, defaecation was seen immediately thereafter and reduction of enterogastric reflux followed closely with continued enteral feeding and exercise. Postoperative defaecation, together with tolerance of solid food intake, may be the best clinical endpoint of POI in humans.[25] As numerous factors convened around the time of clinical resolution of POI (reduced SI wall thickness, carbachol treatment, increased oral food intake and initiation of treadmill exercise), it is impossible to know the impact of each individual factor; in fact, the effect of time itself on POI resolution should not be underestimated either.

Human guidelines underline the importance of a multimodal approach to treatment and prevention of POI,[22 23] and early resumption of oral nutrient intake may limit POI duration and severity in humans.[4 5] Interestingly, gum chewing has a small but significant protective effect, presumably because it acts as sham feeding, promoting GI motility and coordination through stimulation of the cephalic-vagal reflex.[26] A beneficial effect of early postoperative feeding has also been noted in horses following SI surgery.[27] Our patient was offered small handfuls of roughage early and allowed to graze (10 min four times daily) from day 3, as this may act as sham feeding. The importance of nutritional support is often underestimated in adult equine patients. The choice of parenteral (PN) or enteral nutrition depends largely on the likelihood of intestinal malabsorption, and both may be combined to meet energy demands in cases of GI system dysfunction. In the presence of prolonged POI with poor tolerance of enteral feeding, the parenteral route may help counter patient negative energy balance and protein catabolism. Studies of parenteral nutrition in adult horses after SI resection showed reduced catabolic markers but increased catheter scores (swelling or phlebitis[28 29]), which parallels the current case. An overall beneficial effect of PN on outcome has not been established in horses. Monitoring weight loss during the first days after laparotomy is good practice and may help, in conjunction with clinical improvement and (in)tolerance of oral feed intake over this time, to estimate the need for PN.

Although exercise is traditionally thought to promote GI motility, there is no direct evidence for this contention after laparotomy in humans.[30] Early ambulation, however, reduces overall postoperative complication rates, which likely contributes to a favourable outcome in human POI cases.[5 22] For our patient, hand-walking was instituted early, but the greatest improvement occurred after uphill walking on a treadmill. This was devised as it reduced the time needed off intravenous fluids for hand-walking, and perhaps the incline would assist gastric emptying.[31] Whether any positive effect of uphill exercise on GI transit in horses does indeed occur has not been formally addressed, and potential benefits of such physical activity after laparotomy must be weighed against the risks of abdominal wound complications. The incisional site infection (and slight cosmetic hernia) that ensued in our patient were likely to be more related to repeat laparotomy, but we cannot know this for certain.

In conclusion, the current case highlights that equine POI may resolve, even in longstanding cases, if welfare and economic considerations allow continued treatment, and in this respect may mimic prolonged POI in humans. Prospective randomised trials evaluating the efficacy of a multimodal approach to treatment of equine POI, addressing fluid therapy and prokinetic drug use and particularly also early enteral feeding and ambulation, would seem warranted.

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