

Colic: medical treatment and management in horses

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ABSTRACT

Colic is a common condition in horses and all cases require medical management, to a greater or lesser extent, regardless of the underlying cause and whether they additionally undergo surgery. The aims of medical management are to relieve pain; correct and maintain hydration status, including electrolytes and acid-base disturbances; treat endotoxaemia; and treat ileus. This is achieved using combinations of analgesia, fluid therapy, antiendotoxic therapy and motility stimulants. Drugs used to provide analgesia include NSAIDs, α_2 -agonists, opioids and spasmolytics. Fluid therapy can be administered via the enteral route or intravenously once the deficit has been estimated from the clinical signs and clinical pathology data.

An ongoing fluid therapy plan should then be formulated to account for the maintenance requirements and ongoing losses. In addition, any electrolyte and acid-base disturbances can be corrected using electrolyte supplementation of the fluids and specific fluids. Inotropes and vasopressors should be reserved for specific cases that do not respond to fluid therapy alone. Antiendotoxic therapy options include removing the underlying cause, neutralising circulating endotoxin, inhibiting the endotoxin-induced activation of the inflammatory cascade and inhibiting the endotoxin-induced alterations in the coagulation/fibrinolytic systems. Motility stimulants used in cases with ileus include metoclopramide, lidocaine and erythromycin.

Colic (Figure 1) is a common condition in the horse and all cases require medical management, to a greater or lesser extent, regardless of the underlying cause and whether they additionally undergo surgery.

The aims of medically managing colic are to:

- relieve pain

- correct and maintain hydration and electrolyte and/or acid-base abnormalities
- treat endotoxaemia
- treat ileus

Analgesia



Figure 1. Horse showing signs of colic.

Before a specific diagnosis has been reached, the ideal drug has a short duration of action, mild to moderate potency, minimal effect on gastrointestinal motility and minimal effect on the cardiovascular system. In contrast, once a specific diagnosis has been reached, a longer duration of action is often more appropriate and products with additional benefits, such as antiendotoxic effects, are often used. However, none of the following available drugs are ideal and the potential side effects have to be considered in each individual case.

NSAIDs

NSAIDs inhibit cyclo-oxygenase (COX) enzyme and, therefore, the production of prostaglandins and thromboxane. Prostaglandins directly and indirectly stimulate nerve endings – resulting in pain. The side effects associated with NSAID administration in the horse also due to COX inhibition include gastric glandular ulceration, renal papillary necrosis, right dorsal colitis and pancytopenia.

Other potential side effects include delayed recovery of small intestinal epithelial barrier function as prostaglandins stimulate the repair of injured intestine and are critical for the recovery of barrier function.

NSAIDs licensed for treatment of abdominal pain in the horse include:

- metamizole
- flunixin meglumine
- meloxicam

- ketoprofen

?₂-agonists

?₂-agonists are hypnotic sedatives with excellent gastrointestinal analgesic properties. These drugs appear to act by stimulation of central ?₂-adrenoreceptors, which modulates the release of norepinephrine and directly inhibits neuronal firing. Side effects include hyperaesthesia, hypotension, bradycardia, gastrointestinal ileus, sweating, diuresis and ecbolic effects. Thus, they are effective visceral analgesics, but decrease gastrointestinal motility for the duration of the period of sedation and are cardiovascular depressants.

?₂-agonists available for use in the horse include:

- xylazine
- detomidine
- romifidine

Opioids

Opioids act on opioid receptors in the brain, spinal cord, autonomic nervous system, gastrointestinal tract and, peripherally, at terminals of sensory neurones resulting in analgesia. They are classified as pure agonists (such as morphine, pethidine, methadone or fentanyl) or partial agonists (such as butorphanol and buprenorphine).

Side effects include respiratory depression, hypnosis, excitement, mydriasis, locomotor response, bradycardia, pruritus and non-propulsive gastrointestinal spasm. Only butorphanol and pethidine are licensed for the relief of abdominal pain in the horse. Buprenorphine is licensed for the relief of postoperative pain and other drugs are used under the cascade system.

Spasmolytics

Hyoscine butylbromide (N-butylscopolamine) is a spasmolytic agent with particular activity on the smooth muscle of the digestive and urinary systems. It antagonises the actions of acetylcholine at the muscarinic receptor and also has some activity at nicotinic receptors. It is licensed in the UK as an aid in the control of pain associated with simple equine colic, as a diagnostic aid in more severe equine colics and for the control of diarrhoea in horses, particularly when pain or abdominal discomfort is present.

Infusions

Analgesia can also be provided in the form of a constant rate infusion – for example:

- Butorphanol – 12mg/kg/hr.
- Morphine – 0.3mg/kg loading dose, then 0.05mg/kg/hr.
- Lidocaine – 1.3mg/kg over 15 min, then 0.05mg/kg/min.
- Ketamine – 0.4mg/kg/hr to 0.8mg/kg/hr.
- Trifusion – loading doses of lidocaine (1.3mg/kg over 5 min) and morphine (0.1mg/kg IV bolus) followed by infusion of bag one containing lidocaine (3mg/kg/hr) and ketamine (0.6mg/kg/hr) and bag 2 containing morphine (0.025mg/kg/hr).
- Pentafusion – loading doses of lidocaine (1.3mg/kg over 5 min) and morphine (0.1mg/kg IV bolus) followed by infusion of bag 1 containing lidocaine (3mg/kg/hr) and ketamine (0.6mg/kg/hr) and bag 2 containing morphine (0.025mg/kg/hr), detomidine (0.0044mg/kg/hr) and acepromazine (0.0022mg/kg/hr).

No veterinary-licensed opioids are approved for administration by infusion.

Fluid therapy

Aims

The aims of fluid therapy are to restore the circulating volume and improve the cardiac output – which, in turn, will increase oxygen delivery to the tissue and sustain aerobic metabolism – and correct electrolyte and/or acid-base disturbances.

Estimating deficit

Estimation of the fluid deficit in an individual animal is based on the clinical signs and clinical pathology data. The clinical parameters that should be assessed include the heart rate, jugular refill time, mucous membrane colour, capillary refill time, skin turgor, temperature of the extremities and urine output.

The clinical pathology parameters that should ideally be measured include the blood PCV, total protein, creatinine, urea and venous oxygen concentrations, urine-specific gravity and the central venous pressure. However, it should be remembered these should be interpreted in light of the primary condition. For example, pain will also cause the heart rate to increase, while a protein-losing enteropathy may mask any dehydration-associated increase in total protein. A horse will start to show mild signs at 5% dehydration and severe signs at 12% dehydration.

Enteral fluids

Enteral fluids are contraindicated if there is ileus, intestinal obstruction, severe mucosal inflammation, the horse is unable to stand, or if the horse requires rapid, large volume resuscitation. The advantages of enteral fluids are lower cost and the fact the gastrointestinal

mucosa acts as a natural selective barrier – making iatrogenic imbalances less likely, absorption is increased in hypovolaemia and the haemodynamic effect is rapid (evident from 30 minutes).

Thus, enteral fluids are indicated to restore the hydration status and electrolyte balance, or to prevent this from occurring in horses with ongoing losses that are not drinking. In addition, they are indicated to increase the hydration of the gastrointestinal contents and to stimulate intestinal motility via the gastrocolic reflex.

Enteral fluids are administered via a nasogastric or naso-oesophageal tube as a bolus or continuously. Ideally, the fluid administered should be made isotonic through the addition of sodium chloride (4.9g/L of water) and potassium chloride (4.9g/L water).

Potential complications include aspiration, abdominal discomfort, gastric rupture and nasogastric tube-associated complications.

Intravenous fluids

Vascular access



Figure 2. The jugular vein is the only site suitable for high volume resuscitation.

The jugular vein is most commonly used as the site for catheter placement (**Figure 2**) as it is the only site suitable for high volume resuscitation – and it should be remembered both jugular veins can be catheterised if maximum volume resuscitation (about 35L/hr) is required. Alternative sites for catheter placement include the lateral thoracic, cephalic and saphenous veins.

Fluid therapy plan

A fluid therapy plan should be formulated to take into account the current deficit, maintenance requirements (40ml/kg/day to 60ml/kg/day) and ongoing losses – for example, diarrhoea and gastric reflux. The appropriate type of replacement fluid should be selected and the deficit should be replaced over the next few hours (**Figure 3**). Commonly, about half of the deficit is administered and then the horse reassessed to determine the response to therapy by serially measuring various clinical signs and laboratory parameters, with the remaining requirement adjusted as necessary.

Solutions

Available solutions are either crystalloids or colloids. Crystalloids contain water, sodium or glucose, other electrolytes, plus or minus a buffer. The fluid may be hypotonic, isotonic or hypertonic relative to plasma. They distribute between the intravascular (25%) and interstitial compartments (75%) within one hour, thus, 1L of crystalloids results in a 250ml increase in plasma volume.

Distribution into the interstitial compartment is beneficial if the animal is dehydrated as well as hypovolaemic, but it will also promote tissue oedema formation, which may compromise perfusion.

Distribution into the intravascular compartment will result in a decrease in the total protein concentration (through dilution), which, in turn, will decrease the colloid osmotic pressure, thus favouring further fluid loss into the interstitium, which, again, may be beneficial or detrimental depending on the individual case. These factors should all be borne in mind when considering the fluid therapy plan.

Isotonic polyionic solutions are the most commonly used crystalloid solution in equine veterinary practice. Their electrolyte composition is similar to plasma; however, the potassium concentration is too low for maintenance requirements, so 10 milliequivalent potassium chloride; mEqKCl/L to 20mEqKCl/L should be added. Hartmann's solution is most commonly used as it is available in 5L bags. Isotonic saline (0.9% sodium chloride; NaCl) is hypernatraemic and hyperchloraemic relative to plasma and lacks other electrolytes. It is only available in 1L bags and so is primarily used in cases of hyponatraemia and hypochloraemia, rather than for resuscitation or maintenance.

Hypertonic saline (7% NaCl to 7.5% NaCl) is hypertonic relative to plasma, so it initiates movement of water into the intravascular space from the interstitium, resulting in rapid expansion of the circulating volume. The plasma volume expansion achieved is two times to four times that of the infused volume (2ml/kg to 4ml/kg). However, this will result in intracellular dehydration, so it should be followed up with 10L isotonic fluids for every 1L of hypertonic saline administered within 2.5 hours.



Figure 3. Intravenous fluid therapy.

A 5% dextrose is a hypotonic solution, which will replace water without electrolytes, thus, it is only used in animals that are hypernatraemic and hyperchloraemic. It should be used with caution as it can cause hyperglycaemia with rapid administration, which will subsequently result in osmotic diuresis and so further fluid loss.

Colloids contain large sugar or protein molecules and are a mix of large and small molecules. The advantages of colloids over crystalloids are the large molecules improve oncotic pressure and they provide rapid intravascular volume replacement by expanding the plasma volume by 100% to 200% of the volume infused, thus improving microvascular perfusion with less tissue oedema formation. The disadvantages are they are affected by alterations in capillary permeability, do not correct dehydration (interstitial losses) and can have side effects in some animals. Colloids available in the UK include plasma, whole blood and the synthetic colloid gelofusine.

Electrolyte supplementation

Electrolyte concentrations should be measured as the initial correction of the fluid deficit and then appropriate supplementation of the fluids should be initiated and the response monitored.

Acid-base disturbance

The most common acid-base disturbance encountered is metabolic acidosis, which occurs as a consequence of lactic acidosis secondary to hypovolaemia and/or endotoxaemia, or of hyponatraemia secondary to colitis, peritonitis or gastrointestinal torsion.

Metabolic alkalosis occurs as a consequence of hypochloraemia secondary to high volume gastric reflux or of hypoalbuminaemia. Respiratory acidosis may occur as a consequence of hypoventilation and respiratory alkalosis may occur as a consequence of hyperventilation due to pain. Treatment should be aimed at the underlying cause, thus, lactic acidosis should be treated with a large volume of polyionic fluids, hyponatraemia with normal or hypertonic saline, hypochloraemia with normal saline and hypoalbuminaemia with colloids.

Vasopressors and inotropes

Critically ill horses often have disturbances of their cardiovascular system, which can result in inadequate oxygen delivery. For example, endotoxaemia and ischaemia-reperfusion injury may induce capillary damage, allowing fluid and albumin to leak out, causing hypovolaemia and reduced intravascular colloid oncotic pressure and increased interstitial colloid oncotic pressure to draw out more fluid. Sepsis and endotoxaemia are also characterised by vasodilation, which leads to relative hypovolaemia, and endotoxin and sepsis result in cytokine production that cause myocardial depression.

Inotropes and vasopressors can be used to ensure adequate oxygen delivery to the tissues if it is still insufficient following appropriate fluid therapy. Inotropes increase the cardiac output by increasing the stroke volume and evidence suggests dobutamine is the best available drug. Vasopressors increase the blood pressure through arteriolar vasoconstriction, which increases the pressure gradient across a capillary bed, allowing better perfusion. However, it will also increase the resistance to flow, so the dose needs to be titrated carefully to achieve optimum balance between perfusion pressure and flow. Evidence suggests noradrenaline is the first choice drug.

Antiendotoxic therapy

The aims of antiendotoxic therapy are to:

- Remove the underlying cause. This involves surgical correction of intestinal lesions, drainage of peritoneal effusions and antibiotic therapy.
- Neutralise circulating endotoxin. Polymyxin B binds to the lipid A and neutralises endotoxin. Low doses (6,000IU/kg IV q8-12h) can be used systemically without toxicity.

- Inhibit the endotoxin-induced activation of the inflammatory cascade. Options for inhibition of endotoxin-induced inflammation include the NSAID flunixin meglumine, pentoxifylline and lidocaine.
- Modulate the endotoxin-induced alterations in the coagulation system.

Endotoxin-induced inflammatory mediators activate the coagulation system and inhibit the fibrinolytic system. This procoagulant state exhausts the supplies of clotting factors so bleeding diathesis then ensues. Options for therapy include plasma transfusion to replace the supply of clotting factors and low molecular weight heparin to bind to antithrombin III – increasing its affinity for and inhibition of activated clotting factor x.

- Provide circulatory support.

Fluid therapy is an essential part of the treatment of endotoxaemia.

Motility stimulants

Postoperative ileus should be treated with a combination of analgesia, decompression, antiendotoxic therapy, correction of electrolyte imbalances and the following available prokinetic drugs.

Lidocaine

Lidocaine improves gastrointestinal smooth muscle contractility, but the mechanism involved is unknown and also has analgesic and anti-inflammatory properties. It is administered as a loading dose of 1.3mg/kg IV bolus over 5 minutes, followed by 0.05mg/kg/min IV infusion for 24 hours. Side effects include muscle fasciculations, ataxia and seizures.

Metoclopramide

Metoclopramide augments acetylcholine release from cholinergic neurones stimulating motility in the stomach and proximal duodenum. It should be administered as 0.25mg/kg IV in 1L of saline over 30 minutes to 60 minutes or as 0.08mg/kg/hr to 0.14mg/kg/hr infusion. Potential side effects include CNS excitement due to dopaminergic antagonism.

Erythromycin

Erythromycin acts via motilin and/or five hydroxytryptamine receptors. However, the prokinetic response may decrease with repeated use due to receptor down-regulation. It should be administered as 2mg/kg IV qid by slow IV injection. Undesirable side effects include abdominal pain.