Common medical problems in the older foal. Part 2: Diarrhoea

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Diarrhoea is a common condition in the foal, with up to 80% of foals experiencing one episode before six months of age (Traub-Dargatz *et al.,* 1988). This diarrhoea can be the result of normal physiological changes occurring in the foal's gastrointestinal tract, dietary and management factors or infectious agents. The immature foal colon is unable to compensate for pathology of the small intestine and, as a result, most clinical cases in foals are due to changes affecting this area.

CLINICAL PRESENTATION

The clinical presentation of diarrhoea cases can vary depending on the severity of the case. Often the presenting and most life threatening clinical signs are sequelae to the cardiovascular derangements that result from the associated fluid and electrolyte loss within the gastrointestinal tract. Bacterial translocation across the damaged intestinal mucosa may result in clinical signs of endotoxaemia due to reduced systemic vascular resistance and organ hypoperfusion. In contrast, physiological causes of diarrhoea, such as lactase deficiency, may result in mild signs of osmotic diarrhoea, in the absence of any systemic signs.

DIAGNOSTIC APPROACH

As with any case, establishing a comprehensive history of the foal, the mare and of the yard is crucial in establishing differential diagnoses. The age of the foal, history of passive transfer, parasite control and previous disease history are all important, as is information pertaining to any other clinically affected animals from the same environment and husbandry practices in the affected yard. A good history combined with a thorough physical examination is often enough to form a presumptive diagnosis, and is often all that is available at the initiation of treatment.

The physical examination should include assessment of all major body systems, hydration status and should identify any concurrent abnormalities. A haematological profile and, if available, an acute-phase protein profile are useful in determining the aetiology. Initial treatment will involve stabilising cardiovascular and electrolyte aberrations and therefore blood gas analysis, if available, is very helpful.

Faecal samples taken directly from the rectum and placed immediately in selenite broth are useful for specific tests as indicated by the case, such as bacterial culture for *Salmonella,* microscopic evaluation for parasitic stages or ELISA analysis for clostridial toxins or rotavirus isolation.

There are many causes of diarrhoea in the older foal, including infectious and non-infectious causes. Table 1 lists the most common infectious causes of diarrhoea in older foals.

TABLE 1: Infectious causes of diarrhoea in foals	
Type of pathogen	**Pathogen**
Viral	Rotavirus
	Coronavirus
Bacterial	*Rhodococcus equi*
	Lawsonia intracellularis
	Clostridia spp.
	Salmonella spp.
Parasitic	Ascarids
	Cyathostomiasis
	Large strongyles
Protozoal	*Cryptosporidium*

Non-infectious causes include:
- Antibiotic-related diarrhoea
- lactose insufficiency
- dietary hypersensitivity
- errors of feeding, e.g. incorrect formulation, volume, concentration

VIRAL DIARRHOEA

Cells at the tips of the villi are destroyed, the gut loses absorptive capacity and lactase is destroyed. Maldigestion and malabsorption occur, often combined with increased gut secretion. As the cells in the crypts are not affected malabsorption is usually a transient change. Diarrhoea is a result of the osmotic gradient drawing water into the gut lumen. There is no protein leakage.

Rotavirus

Rotavirus infection is the most common cause of viral diarrhoea in the foal. Various serotypes have been identified, all of which belong to group A. Foals are infected via oro-faecal transmission, often at 1–8

weeks of age, although some older foals can be affected and may show less severe clinical signs. The virus is highly contagious, often occurring in outbreaks, and can persist in the environment for several months. Clinical signs vary from depression and failure to suck to more severe disease. Initially foals will be pyrexic. Diarrhoea is often a green to grey colour and is non fetid.

Diagnosis

Diagnosis is made by demonstrating viral particles in faeces using a commercial ELISA test, latex agglutination or electron microscopy. Electron microscopy is the gold standard but it is not routinely available and results take several days. Virus isolation is difficult as rotaviruses are difficult to grow in cell culture.

Treatment

Treatment consists of maintaining hydration, oral electrolyte solutions may be sufficient, and anti-ulcer medication. Hyperimmune plasma is available and can be administered prophylactically intravenously to at-risk foals. Colostral antibodies are not protective alone as cellular immunity is thought to be an important aspect of recovery.

These foals do best being maintained on mares' milk as this contains locally-protective IgM.

Prevention

Inactivated vaccines have been developed for use in mares during the eighth, ninth and tenth months of gestation, and are shown to increase antibody levels in mares and their foals for up to 90 days.

Coronavirus and adenovirus

Coronavirus and adenovirus infections are much rarer in the foal, although there are case reports in the literature of these viruses as causes of clinical diarrhoea in immune-suppressed animals.

BACTERIAL ENTERITIS

With the exception of *E. coli* infection, in bacterial infections such as *Salmonella* and *Clostridia* the bacteria invade the mucosa and submucosa causing extensive damage. Cells at the base of the crypts are more severely affected, hence the changes are more persistent. Polymorph leukocyte invasion and inflammation of the deeper layers of the intestinal wall occur. There is a loss of absorptive and secretory function, leakage of plasma proteins and intestinal spasms. Diarrhoea is often profuse, fetid and haemorrhagic.

E. coli

E. coli is one of the most common causes of sepsis in foals but it is not usually associated with diarrhoea, unlike in farm animals. There is no relationship between its presence in faeces and the incidence of diarrhoea. Diarrhoea may be caused by the production of an enterotoxin resulting in hypersecretion or direct invasion of the mucosa. *E. coli* is commonly cultured in faeces and therefore diagnosis should be based on PCR demonstrating toxin production. If *E. coli* septicaemia is present aggressive treatment may be required. Clinical signs vary from mild transient diarrhoea to more severe signs.

Salmonellosis

All reported cases of *Salmonella* infection are from the species *Salmonella enterica,* which contains six subspecies and over 2000 serovars. The prevalence of each serovar varies between studies: *S. typhimurium* and *S. enteritidis* are the most prevalent in Europe. *S. anatum* and *S. agona* have been found to be more prevalent in Australia and Kentucky respectively. The primary route of infection is thought to be oral transmission, through contaminated feed and water, faeces of infected animals or the environment. *Salmonella* can persist in the environment in dry faeces for several years and surface water for four months. It can cause diarrhoea in foals of any age but is most commonly identified in those between one and four months of age. Nosocomial infection is common.

Clinical signs

Clinical signs vary but often include pyrexia, acute fetid diarrhoea and hypotensive shock. Signs of septicaemia may precede the onset of diarrhoea. Signs of localised infections such as synovitis, osteomyelitis, uveitis or physitis may be present. Haematology often identifies a substantial neutropenia, with toxic change and a left shift.

Diagnosis

As shedding is intermittent in faeces, it is recommended that bacterial culture and isolation from five sequential faecal samples be taken; it will be several days before the results are available. Faecal culture has poor sensitivity, approximately 55%, for one sample, which is improved when using selenite-enriched broth. Culture allows typing of isolates, which is important in tracing outbreaks of disease. Rectal mucosal biopsies are more sensitive than faecal isolation but are more invasive. A faecal PCR, which detects the histadine transport operon gene of *S. typhimurium,* is available. This provides quicker results and has a greater sensitivity than faecal culture. Blood culture is also used in septicaemic foals.

Post-mortem examination reveals a diffuse haemorrhagic or fibrinous inflammation of the large colon. Pseudomembranes may be present. Enlarged mesenteric lymph nodes may be haemorrhagic or oedematous. Similar changes may be seen in the stomach and small intestine. Histological changes include mononuclear cell infiltrate, and occlusion of the capillaries in the *lamina propria* by fibrin. Foci of hepatic necrosis may be present.

Treatment

The use of antimicrobials is controversial, as they do appear to increase the risk of faecal shedding of *Salmonella*, and affect the normal gut flora. Antibiotics are more indicated in neutropenic horses at risk of bacterial translocation across the damaged epithelium. The ideal antibiotic would be an antibiotic such as enrofloxacin that is bactericidal, affects Gram -ve bacteria, has intracellular penetration, minimal resistance and minimal effect on the commensal flora. However, enrofloxacin is contraindicated in foals due to its effects on cartilage maturation. If antibiotics are deemed necessary, broad-spectrum coverage should be provided. Multi-drug resistance is a particular problem with *S. typhimurium* DT 104, particularly in other species, e.g. humans.

Clostridiosis

C. perfringens type A colonises the intestinal tract of neonatal foals within the first few days of life and as a result is normally cultured in the faeces. *C. perfringens* type C and type A can be associated with diarrhoea in older foals. Colostrum has been associated with an increased risk of clinical signs: it is postulated that trypsin inhibitors, present in the dam's colostrum to protect IgG, may allow toxins to persist.

Clinical signs and diagnosis

Clinical signs include acute abdominal pain, fever and haemorrhagic diarrhoea (Fig. 1) and are associated with a mortality. *C. difficile* can be cultured in healthy foals, unlike healthy adult horses, but it can also be a primary pathogen, being less associated with previous antibiotic therapy than in adults.

Serum biochemical changes are highly variable. Abdominocentesis may yield an exudate if severe enteritis is present. Gas and fluid distension of the small and large intestine may be visible on

Fig. 1: *Haemorrhagic diarrhoea in a foal caused by Clostridium perfringens. (Courtesy of Imogen Johns BVSc DACVIM MRCVS)*

ultrasonography and radiography. Rectal swabs may reveal many Gram +ve rods. Bacterial culture and toxin isolation from faecal samples are required.

Treatment

In foals with *C. perfringens* symptomatic treatment should be provided. Analgesics such as butorphanol combined with anti-ulcer medication may be necessary. DTO smectite and probiotics containing *Saccharomyces* have all been described. Broad-spectrum antibiotics, e.g. penicillin in combination with amikacin or cephalosporins and metronidazole, should be given and milk should be withheld as malabsorption results in osmotic diarrhoea. Oral antitoxins to type C and D have been described but there is no evidence to support a beneficial effect of this. Hyperimmune plasma is available.

Rhodococcus equi

R. equi is an aerobic Gram +ve bacillus and saprophytic soil inhabitant. It is an intracellular bacterium which resides in macrophages, resulting in necrosis of lymphatic tissue within Peyer's patches of the gastrointestinal tract. Infection can result in bronchopneumonia or enteric infection with septicaemic forms sequestering the organism into joints, bone, eyes or the liver. Affected foals are usually 2-3 months of age and often present with lethargy, depression, fever, suppurative colitis or colic. It is rare to get alimentary signs without pulmonary involvement. Approximately 50% of those with bronchopneumonia also have intestinal pathology.

Diagnosis

The organism is easily isolated from diarrhoeic faeces. Culture and identification via PCR is the gold standard for diagnosis. Serological tests, including western blots, ELISAs and haemolysis inhibition, are available.

Treatment

Macrolides and rifampicin (5 mg/kg q 12h PO) act synergistically to provide good penetration of abscesses and cells. Erythromycin can itself result in diarrhoea and hyperthermia. If this occurs, a gentamicin and rifampicin combination may have to be used. Various erythromycin preparations are available, but erythromycin estolate has the best absorption, fewest side effects and a lower dosing frequency. Azithromycin has the advantage that it only needs to be given once a day and clarithromycin only twice a day compared to erythromycin which requires three or four times daily dosing. Clarithromycin has been associated with better treatment outcomes.

Plasma has been given prophylactically on endemic farms and hyperimmune preparations are available. Vaccinating mares has not been effective in preventing disease and recent research looking at foal vaccines, using killed bacterin, has been disappointing. Vaccination was more successful using

non-attenuated organisms orally, which results in increased IgG levels.

Lawsonia intracellularis

Proliferative enteropathy caused by *Lawsonia*

Fig. 2: *A six-month-old Percheron foal presented with a three-week history of worsening diarrhoea. (Photograph courtesy of Keith Poulsen DVM DACVIM.)*

Figs. 3 and 4: *A percutaneous abdominal ultrasonographic scan of the above foal. Note the thickened walls of the duodenum (normal <0.3cm).*

Fig. 4.

Fig. 5: *Post-mortem specimen of above foal, showing thickened oedematous haemorrhagic small intestine.*

intracellularis causes diarrhoea in foals of 4-7 months of age (Fig. 2). Typical lesions are seen along the jejunum and ileum and can be seen ultrasonographically as a profoundly thickened small intestinal wall (Figs. 3 and 4), leading to a decrease in gut lumen.

Clinical signs include diarrhoea, colic, anorexia, weight loss, a failure to thrive and ventral oedema. Hypoalbuminaemia with neutrophilia are characteristic laboratory findings and confirmation of the diagnosis can be made via faecal PCR or identification of the organism within the intestinal mucosal crypt cells at biopsy.

The treatment of choice is a prolonged course of oxytetracycline. The use of macrolides in conjunction with rifampicin, for their intracellular penetration, has also been described.

PARASITIC DIARRHOEA
Migrating larvae result in inflammation and infiltration of the intestinal wall; some also cause vascular damage and motility disturbances.

Large strongyles
Traditionally the large strongyles were associated with colic and diarrhoea, but interval worming strategies have dramatically reduced their incidence. *S. vulgaris,* followed by *S. edentatus* and *S. equinus,* is the most important large strongyle implicated in gastrointestinal tract (GIT) disease. Ingestion of a large number of larvae results in acute strongylosis which usually presents as colic. Chronic infection is more likely to manifest as debilitation and possibly diarrhoea.

Cyathostomiasis
Diarrhoea is the result of fourth-stage larval migration through the intestinal mucosa and submucosa, resulting in increased secretion and albumin loss, causing oedema, reduced absorption and infiltration of inflammatory mediators. Cyathostomes also recruit a mononuclear inflammation of the *lamina propria* and a chronic granulomatous colitis has been described. Cyathostomes are often associated with chronic diarrhoea although they can also occur in acute cases. Anthelmintic administration in the presence of a heavy encysted burden may cause rapid larval death and acute inflammation.

Young horses are most commonly affected and present with weight loss, reduced or normal appetite, ill thrift, intermittent fever and diarrhoea. These occur on the re-emergence of hypobiotic larvae in the large intestinal mucosa, in late winter and early spring.

Diagnosis
As disease is caused by the larval stages of the worm, faecal egg counts are not helpful. Haematology often

reveals a neutrophilic leukocytosis and eosinophilia. Biochemistry may reveal a profound hypo-albuminaemia and hyperglobulinaemia. Molecular tools for identification of larval and egg stages are being developed. Other research is aimed at developing methods to allow early detection of anthelmintic resistant genotypes. ELISA experiments using serum from experimentally infected ponies and an endemically infected population have shown increased levels of larva-specific antibodies (IgG(T)). The levels of the antibody response correlated well with the mucosal burden (Matthews *et al.,* 2004).

Treatment
Specific treatment includes either a five-day course of fenbendazole or a dose of moxidectin, although this should be avoided in foals less than four months of age. Corticosteroids are often administered concurrently to reduce the inflammatory reaction in the intestinal mucosa, which has been shown to be more severe with the five-day fenbendazole treatment. Non-steroidal anti-inflammatory drugs, such as flunixin meglumine, have the advantage of being less immunosuppressive.

Ascaridiasis
Ascaridiasis is reported to affect 30-60% of foals. Embryonated eggs ingested from the environment hatch in the GIT and then undergo hepato-pulmonary migration. The pre-patent period is ten weeks.

Clinical disease can manifest as respiratory infection or intestinal damage resulting in weight loss, diarrhoea and occasionally colic. Ivermectin, fenbendazole and pyrantel are all effective and routine worming before six weeks of age will reduce pasture contamination. In the past few years, widespread resistance of *Parascaris equorum* to ivermectin has been identified, therefore this may not be the most effective treatment on studs with a history of resistance.

Cryptosporidiosis
Cryptosporidium parvum has been associated with sporadic cases of malabsorptive diarrhoea in foals. It physically occupies the absorptive surface of the distal small intestine. Most affected foals are 14-21 days old. Immunocompromised foals may be more susceptible. Diagnosis is based on the identification of oocytes following acid-fast staining of faecal samples, electron microscopy, flow cytometry or immunofluorescence assay. This is a contagious and zoonotic organism.

LACTOSE INTOLERANCE
Any condition which results in a loss of the small intestinal mucosal brush border may cause secondary lactase deficiency and subsequent lactose intolerance. This results in physiological diarrhoea due to the osmotic gradient of lactose within the intestinal lumen, drawing fluid in. Affected foals are often lethargic and show a failure to thrive or have weight loss. They may have a history of persistent diarrhoea with recurrent colic from a young age. The diagnosis can be confirmed by a lactose tolerance test but diagnosis is often made by the response to treatment, supplementing with the lactase enzyme. Early weaning is necessary in these cases.

SYMPTOMATIC TREATMENT
It is usually necessary to initiate treatment in the absence of diagnostic information. The priorities include correcting fluid and electrolyte disturbances, controlling endotoxaemia, reducing intestinal inflammation, promoting mucosal repair and encouraging re-establishment of the normal intestinal flora.

Fluids
Fluid deficits should be corrected immediately; often a polyionic crystalloid solution is best. Deficits should be calculated based on clinical dehydration and body weight, and continually reassessed and adjusted as necessary. Once fluid deficits have been rectified, persisting metabolic acidosis can be corrected as necessary using bicarbonate added to the fluids, guided by blood gas measurements.

If the damage to the intestine is severe it is best to withhold milk, to reduce any exacerbation of the diarrhoea via an osmotic effect. However in mild cases the protective immunity provided by the IgA has been advocated. If the absorptive capacity of the intestine is deemed to be normal, enteral fluids would be a cheaper option, but can be labour intensive in the field situation.

If treatment is ongoing it will be necessary to supplement these fluids with glucose in the form of dextrose, to meet the higher-than-normal energy requirements of the sick, catabolic foal. Other electrolyte supplementation may be required, as determined by biochemistry analysis, and should be administered slowly to prevent side effects such as cardiac arrhythmias with potassium supplementation, or central pontine myelinolysis with sodium.

Colloids provide additional oncotic support, especially in hypoproteinaemic foals, which may otherwise sequester fluid into the extravascular space. In this situation plasma would also provide valuable anti-inflammatory mediators and proteins.

Anti-endotoxic therapy
Endotoxaemia results when the body's defence mechanisms are compromised and lipopolysaccharides (LPS) from Gram -ve bacteria are absorbed into the intestine. In the normal horse there are many of these Gram -ve bacteria in the large colon. One of the effects of endotoxin is peripheral vasodilation, which results in decreased systemic vascular resistance and relative hypovolaemia.

In addition to fluid therapy, hyperimmune plasma containing anti-lipid A antibodies, which bind LPS and prevent the initiation of the inflammatory cascade, has been shown to be of clinical benefit to adult horses. In foals these preparations have been shown experimentally to cause a worsening in clinical signs and increased levels of IL-6 and TNF and should therefore be used with caution (Durando et al., 1994).

Di-tri-octahedral smectite is a hydrated alumina-magnesium silicate which is administered to absorb exotoxins and endotoxins. It has been shown to bind toxins in vitro, possibly due to its negative charge which attracts positively-charged exotoxins and endotoxins. Polymixin B (PMB) is a bactericidal antibiotic which targets Gram -ve organisms; at lower doses it binds to the lipid A of LPS. Its efficacy has been demonstrated in experimentally-induced endotoxaemia but this may be reduced once LPS exposure has already occurred.

Intestinal protectants

Anti-ulcer therapy is important to reduce the incidence of gastric ulcers. Omeprazole is the drug of choice, although it is expensive. Sucralfate or an H2 antagonist, such as ranitidine or cimetidine are cheaper alternatives but are less efficacious. The use of bismuth salicylate to decrease inflammation and secretion in the large colon has been described. Bismuth coats the mucosa whilst the salicylate has anti-prostaglandin effects. A relatively new commercial preparation containing montmorillonite is available (Diarsanyl®, CEVA Animal Health), which claims to be much more absorbent than other available formulations.

Sucralfate has been advocated for gastric-acid-related diseases, but its efficacy in the large colon is questionable. Misoprostol has been shown to enhance intestinal mucosal healing in models of colitis in the horse and other species; this has not been confirmed clinically. Systemic absorption acts to re-establish blood flow and mucus production.

Anti-inflammatories

Many mediators have been implicated in the inflammatory response of the intestinal cells. NF-ÎB is the start of a common signalling pathway that activates genes encoding for pro-inflammatory cytokines, including TNF·, IL1 and IL6. Non-steroidal anti-inflammatory drugs (NSAIDs) inhibit cyclo-oxygenase (COX) activity and subsequent production of reactive oxygen metabolites. However other prostaglandins are cytoprotective and important for mucosal repair, therefore caution must be taken to avoid further mucosal damage. Flunixin is currently the NSAID of choice, although the advantage of more selective COX inhibition is being investigated.

Antibiotics

The use of antibiotics in treating the diarrhoeic patient is controversial. There is sufficient documented evidence of an adverse effect of their use, however they may be indicated if an animal is severely neutropenic or septicaemic. In these cases broad-spectrum drugs should be administered until culture and sensitivity results are available. The aim of this therapy is to provide broad-spectrum coverage to reduce bacterial translocation across the damaged intestinal wall, not to target the specific organism causing the diarrhoea. Those antibiotics which specifically target the bacterial wall, e.g. penicillins, may in theory result in an increased release of LPS and worsening of clinical disease.

Probiotics

Probiotics are used to restore the gastrointestinal flora when disease is associated with a disturbance. Products containing bacteria, including Bifidobacterium spp., Lactobacillus spp. and Enterobacter spp., and yeasts such as Saccharomyces have been developed. Concerns over their use include the lack of evidence of a clinical benefit, no information on required dosing regimens and inaccurate label descriptions. Transfaunation has been used to re-establish gut microflora by transferring the caecal contents of a healthy horse to restore the microflora of another.

Anthelmintics

An appropriate worming program, to eliminate pre-patent worm burdens, is important in foals. Moxidectin is not recommended in foals less than four months old, and should therefore be avoided. Ivermectin, fenbendazole or pyrantel are the drugs of choice, according to the likely worm burden and resistance history on the farm.

Motility modifiers

If no infectious cause is suspected, intestinal motility can be reduced temporarily using codeine phosphate. As 75% of cases of diarrhoea are thought to be idiopathic this is a useful treatment in many cases. However, if it is used in the presence of endotoxin, it can facilitate increased absorption and perpetuate disease.

CONTROL

Many of the causes of diarrhoea in the foal are infectious. Good biosecurity is essential to minimise the transmission of disease to other stock on the premises and to prevent disease being transferred from the premises to other establishments.

Husbandry practices should be optimised to reduce the environmental contamination and opportunity for transfer between groups:

- affected foals should be isolated immediately when clinical signs are seen
- affected foals should be handled, ideally by separate personnel wearing appropriate barrier clothing
- other in-contact animals should be kept separately, and rectal temperatures monitored daily to identify and separate any further cases as soon as possible
- housing stock in groups of similar ages should reduce the incidence of disease, by preventing exposure of naïve animals
- equipment and housing should be cleaned and then disinfected thoroughly after removal of the affected animals.

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These multiple choice questions are based on the above text. Answers appear on page 79.

1. **Which of the following statements is NOT correct with regard to rotaviral diarrhoea in the foal:**
   a. Rotavirus is very contagious.
   b. Electron microscopy is the gold standard diagnostic technique.
   c. Rotaviruses are easy to grow in cell culture.
   d. Cellular immunity is thought to be important in the immune response to infection.
   e. Inactivated vaccines are available for use in the final trimester of gestation.

2. **The most prevalent Salmonella serovars in the UK are:**
   a. *S. typhimurium* and *S. agona*
   b. *S. anatum* and *S. agona*
   c. *S. anatum* and *S. enteritidis*
   d. *S. typhimurium* and *S. enteritidis*
   e. *S. typhimurium* and *S. anatum*

3. **Which of the following is NOT correct in foals affected with Rhodococcus equi:**
   a. Approximately 30% of foals with bronchopneumonia attributable to *R. equi* infection have intestinal pathology.
   b. *R. equi* is easily isolated from faeces of infected animals.
   c. Of the available erythromycin formulations erythromycin estolate has the best oral absorption.
   d. Azithromycin is dosed less frequently than clarithromycin and erythromycin.
   e. Hyperimmune plasma can be given prophylactically where the disease is endemic.

4. **Which of the following is NOT true with regard to proliferative enteropathy in the foal due to Lawsonia intracellularis:**
   a. Ultrasonography is very useful in the diagnosis of this condition.
   b. Hypoalbuminaemia is a consistent finding.
   c. Diagnosis can be confirmed by faecal PCR.
   d. The treatment of choice is a prolonged course of procaine penicillin.
   e. Macrolides and rifampin may also be used to treat the infection.

5. **In parasitic diarrhoea which of the following statements is CORRECT:**
   a. In cyathostomiasis, diarrhoea is a result of migration of encysted third stage larvae.
   b. Cyathostomes recruit a neutrophilic inflammation of the intestinal serosa.
   c. An IgM test is being developed as a potential marker of larval burden.
   d. A five-day course of fenbendazole or a single dose of moxidectin is required to treat the encysted larvae.
   e. Anti-inflammatory drugs are contraindicated.