

Foal diseases

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Foal diseases

Most conditions in foals require intensive, long-term treatment undertaken in specialist facilities, with varying success. Secondary complications and septicaemia are a common sequel to many foal conditions, resulting in death particularly in the first weeks of life. Often quite simple measures can be very effective in avoiding disease in the first few weeks of life, such as ensuring foals receive **adequate colostrum**.

Failure of passive transfer

All neonatal equids are born without circulating immunoglobulin and initial immune protection is passed from the mare via the colostrum through intestinal absorption. Antibodies are absorbed from the intestine for the first 12–18 hours after birth. **Failure of passive transfer (FPT)** is the result of insufficient absorption of immunoglobulin from colostrum. Without circulating immunoglobulin, foals are vulnerable to a wide range of secondary opportunistic infections which can be severe or even fatal.

Causes

- Delayed colostrum intake
- Leakage of colostrum from the udder before the foal can drink
- The mare not allowing the foal to drink, e.g. in cases of mastitis or if a maiden mare rejects the foal
- Poor colostrum quality

Following FPT a foal is likely to develop a bacteraemia (bacteria in the bloodstream) leading to: depression, weakness, respiratory distress, inappetence, diarrhoea, congested/septic mucous membranes and joint ill.

Ensure all foals receive their first meal of colostrum within the first 2–4 hours of birth.

If a foal cannot drink, either support the foal every hour to allow suckling, or milk the mare and feed the foal. If there is insufficient colostrum production, supplement with milk from another mare.

Foal diarrhoea

Diarrhoea (scour) is common in foals. In most cases it is self-limiting and resolves with **fluid therapy** rather than medication. Foals become dehydrated rapidly; correct the fluid deficit if present. If a foal stops feeding, more aggressive treatment may be necessary as the foal will soon become dehydrated and hypoglycaemic.

Nutritional diarrhoea

Changes in milk composition, excessive milk intake, or ingestion of foreign material can cause mild and transient nutritional diarrhoea.

Treatment

Allow the foal to suckle little and often to prevent excessive intake, keep standing areas clean and pick up faeces. Avoid antibiotics as this will disrupt the balance of intestinal bacteria. Offer oral glucose/electrolyte solutions in drinking water.

Infectious diarrhoea

Knowledge of the most common pathogens at certain age groups will aid diagnosis and effective treatment.

Pathogens which can affect all age groups of foals

Clostridium difficile

Clostridium perfringens types A and C

Salmonella spp.

These bacterial infections can be severe in foals less than 1 week old.

Pathogens that can affect foals from 1 week to 2 months

Rotavirus (Diarrhoea is often benign but has the potential to progress to life-threatening disease.)

Cryptosporidium (Frequently causes mild, self-limiting diarrhoea. Crypto is most common within the first 3–4 weeks. Foals over 6 months can develop chronic diarrhoea.)

Pathogens that can affect foals older than 2 months

Large parasite burdens have the potential to cause diarrhoea in all foals older than 1 or 2 months.

Cyathostomiasis should be considered as a cause of diarrhoea in foals from 2 months of age onwards.

Lawsonia intracellularis causes proliferative enteropathy with diarrhoea and hypoproteinaemia in weanlings from 3 months of age.

Diagnosis

Without access to laboratory facilities, diagnosis of the aetiology of foal diarrhoea can be difficult. *Cryptosporidia* and cyathostomes can be diagnosed by microscopic faecal examination, although encysted cyathostomes may not be producing eggs. An acid fast stain must be used to detect *cryptosporidium* in a faecal smear. Local laboratories may have access to further testing facilities, e.g. faecal culture testing for bacterial diarrhoea. Diagnosis based only on clinical signs is likely to be inaccurate; however, profuse watery faeces with blood, mucus and a foul smell is typical of bacterial diarrhoea.

Treatment

The most important aspect of the treatment of foal diarrhoea is to **maintain hydration** (see Fluid therapy on page 504). If systemic illness is severe use a penicillin (20,000 IU/kg) and gentamicin (3.5 mg/kg; Crisman et al. 1997) combination, trimethoprim-sulphonamide is also acceptable. Antibiotic use in non-bacterial diarrhoea can make the symptoms worse. Good nursing care is essential. To reduce the helminth burden keep the environment clean and pick up faeces daily. Determine the frequency of anthelmintic use with a FEC of mare and foal. *Salmonella* and *cryptosporidium* are zoonotic; strict hygiene and isolation are essential. Unfortunately,

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the prognosis for severe systemic illness is poor as death can result from enterotoxaemia, dehydration and circulatory collapse.

Fluid therapy in foals

Successful fluid therapy in foals relies upon early diagnosis of hypovolaemia and rapid fluid replacement. Clinical signs of hypovolaemia are less obvious in foals than in adult equids. Moreover, foals are less able to adapt physiologically to hypovolaemia so can deteriorate quickly. The clinical signs of hypovolaemia include increased heart rate, increased respiratory rate, reduced pulse strength, reduced jugular filling, and cold extremities (distal limbs, ears, and nose). Attempts should be made to maintain hydration, using oral electrolytes, in foals with diarrhoea. A small stomach tube can be used for administration in foals that are not drinking. Check that the tube is in the oesophagus by feeling the tube in the neck.

Intravenous fluid therapy

When working in a field clinic, maintaining foals on intravenous fluids can be problematic; if left unattended risks include a fluid overdose or thromboembolitis. **Fluid boluses** can be given quickly with minimal complications.

Foals are usually recumbent with hypovolaemia, and almost always lethargic/dull.

Administer 0.5 litres (15-kg foal) or 1 litre (50-kg foal) of isotonic electrolyte solution via a 16–18 gauge jugular catheter. In cases of diarrhoea use Lactated ringer's/Hartmann's solution, as saline is mildly acidifying.

Wait 15 minutes and monitor the response.

- If the bolus of fluids is sufficient, then the foal will become brighter and attempt to get up.
- Urination is a useful indication of improvement; however, foals will continue to urinate in the face of hypovolaemia/dehydration as the kidneys are immature.
- Monitor clinical signs.
- If the foal remains recumbent and depressed, a second bolus can be given as above (no more than three boluses should be given).

Pulmonary oedema is a rare complication which results from over-hydration. Listen to the thorax with a stethoscope before, during and after fluid therapy. Fine crackles will be heard across the lung fields if pulmonary oedema develops, and a frothy watery discharge can be seen at the nostrils. If this occurs, fluid therapy should be stopped and frusemide (0.25–1 mg/kg IV) administered.

Gastric ulceration

The aetiology of gastric ulceration in foals differs from that in adults. In foals less than thirty days old the predominant cause is reduced perfusion of the mucosa, particularly during illnesses such as sepsis. Neonatal foals are at significant risk of developing **perforating** peptic ulcers, until they are several weeks old because their gastric mucosa is not developed to full thickness at birth. Weaning, intermittent starvation, NSAID therapy, prolonged transportation and other stressful factors have also been implicated in foal gastric ulcer aetiology.

Clinical signs

- May be asymptomatic (clinical signs become apparent when the ulceration is widespread or severe)
- Reduced appetite
- Diarrhoea (a common sign in younger foals)
- Teeth grinding and hypersalivation
- Dorsal recumbency with abdominal pain
- Differential diagnoses include septicaemia, peritonitis or oesophageal obstruction.

Treatment

Treat concurrent illness, avoid the use of NSAIDs. Gastric ulceration can be treated or prevented with oral antacids and gastric protectants.

Proton pump inhibitor – omeprazole 1 mg/kg PO every 24 hours

Histamine receptor antagonist – cimetidine/ranitidine 6–8 mg/kg PO every 8 hours

Gastric protectant – sucralfate 30 mg/kg PO every 6–8 hours

Colic in foals

Colic can be quite common in neonates, and clinical signs of abdominal pain can be slightly different from those shown by adult equids.

Clinical signs

Rolling, pawing the ground, abnormal position when recumbent (lying on back), abdominal distension, teeth grinding, abdominal straining (arched back, raised tail, attempting to defecate), depression, not suckling, increased heart and respiratory rate.

Diagnosis

Meconium impaction is a common cause of colic in the newborn foal; the meconium is usually passed in the first 24 hours. If this does not occur an impaction develops; this can be diagnosed by careful rectal digital palpation.

Other less common causes of colic include the following:

- Gastroduodenal ulceration
- Ileus Secondary to gastrointestinal (GI) hypoxia associated with dystocia (foaling problems) and septicaemia
- Intussusception Euthanasia is indicated on welfare grounds. Ultrasound is used for diagnosis, and surgical correction is the only treatment option. Neither of these is feasible in the working equid context.
- Intestinal volvulus As in adult equids, this is only correctable with immediate surgery as there is severe ischaemic gut injury. This surgery is not possible in field conditions; therefore, euthanasia is indicated on welfare grounds.

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Treatment

To treat a meconium impaction, use an enema to soften the faeces. Insert a small catheter or feeding tube into the rectum to deliver 50 ml of warm soapy water or warm water mixed with rectal lubricant. Analgesics are essential; administer Flunixin meglumine 0.5–1.0 mg/kg IV every 24–36 hours, but use sparingly due to ulcerogenic effect. Alternatively, administer ketoprofen 1–2 mg/kg IV every 24 hours. If treating with NSAIDs, consider concurrent omeprazole treatment to reduce ulcerogenic side effects. To treat ileus, administer pain control and ensure that the foal continues to feed.

Cleft palate

This is a congenital condition where the palate has not fused, leaving an opening between the oral cavity and the nasal cavity. Aspiration pneumonia is a sequelae as the foal is unable to swallow effectively. Assess the size of the defect and the foal's body condition to determine whether euthanasia is necessary.

Clinical signs

Milk can be seen flowing from the foal's nostrils when it is feeding.

Treatment

If the fissure is small the animal may learn to cope and reach maturity. However, if the growth rate is poor, or secondary aspiration pneumonia develops, recommend euthanasia.

Septicaemia

Septicaemia, a systemic inflammatory condition in response to infection, is a major cause of equine neonatal morbidity and mortality. The presence of bacteria in the blood stream is termed 'bacteraemia'. Gram-negative bacteria are the predominant causal organisms. There is a positive correlation between FPT and septicaemia in foals (Robinson et al. 1993).

Clinical signs

- The foal is depressed, not sucking (the mare's udder remains distended).
- Body temperature is not an accurate indicator.
- Lung sounds may still be normal despite major pathology.
- Diarrhoea is common.
- Sudden onset lameness (joint ill) occurs.

Diagnosis

Blood culture is the optimal diagnosis. Take blood aseptically from a surgically-prepared site before beginning any antibiotic therapy. In field situations this may not be possible, so treat according to clinical signs.

Treatment

- Penicillin 20,000 IU/kg plus gentamicin 3.5 mg/kg every 12 hours. (The dose for foals is lower as gentamicin (aminoglycoside) toxicity is a risk in immature kidneys.)

- The treatment duration depends on the clinical response but may be as long as 2 weeks.
- Administer low-dose flunixin meglumine (concurrent omeprazole will reduce risk of gastric ulcers).
- Provide instructions for owners to manage the nursing care for sick foals. Foals should be kept in a clean, warm environment and have access to milk meals every hour (hand-feed weak foals).

Rhodococcus equi

Rhodococcus equi causes a severe bacterial pneumonia in foals. Clinical signs are slowly progressive, and respiratory difficulties may not become obvious until much of the lung is affected by a suppurative pneumonia. The combination of erythromycin (25 mg/kg, PO QID) and rifampin (510 mg/kg PO BID) is effective treatment. The survival rate is high with appropriate therapy.

Joint ill

Joint ill, or synovitis, usually presents as a polyarticular septic arthritis, secondary to systemic infection/septicaemia.

Clinical signs

- Joint enlargement, with heat and pain on palpation/manipulation
- Joint effusion, distension, or thickening of capsule
- Reluctance to move with varying lameness
- Depression, inappetance and fever

Treatment

Use a combination of drugs such as penicillin 20,000 IU/kg IM and gentamicin 3.5 mg/kg. Irrigation of joints may be necessary (see Flushing an infected joint in Section 14.7). If the joint has become damaged the prognosis for work is poor.

Angular limb deformities

Deviations of the limbs medially (varus) or laterally (valgus) (Figure 18.1.1) from the long axis is relatively common in foals. Foals may be born with a mild degree of angular deformity (3–5°) which corrects itself by the time the foal is a 1-year-old. The young growing foal is responsive to interventions. Once the physes have closed (6–24 months depending on the joint) the angulation of the limb cannot be altered by manipulating the growth direction of the limb.

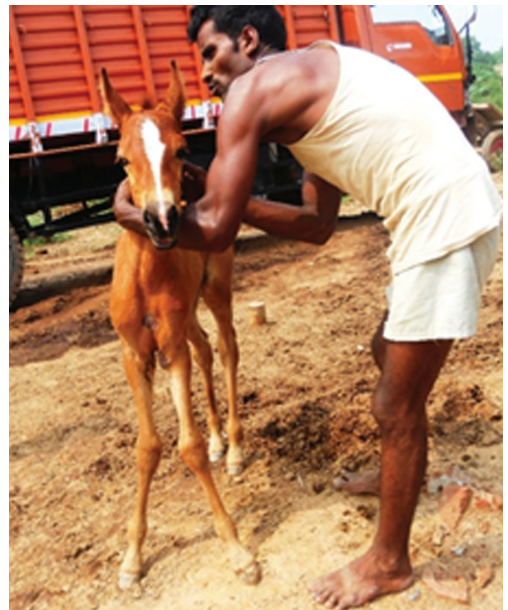


Figure 18.1.1 A foal with valgus angular limb deformity. With restricted exercise this foal showed improvement within 8 days.

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Treatment

In the majority of cases, minimal interventions are required to correct angulations.

- Restrict exercise. (Provide a small patch of land for free exercise only.)
- Shoe-extensions can be employed successfully. (Plastic can be glued to the side of the hoof extending for approximately 3 cm, e.g. apply dorsomedial hoof extensions to correct carpal valgus.)
- Recommend euthanasia in severe cases that show no sign of improvement over the course of several weeks.

Flexural limb deformities

Flexural limb deformities limit the foal's ability to extend a limb to its full extent, and can affect the pastern, fetlock and/or carpus (Figure 18.1.2). The condition may be congenital or acquired, arising secondary to reduced weight bearing in the limb as a result of lameness.

Treatment

- Mild conditions resolve spontaneously.
- Rest in a small enclosure (the size of a stall or stable) provided that the foal is able to stand and suck.
- Straighten and massage the limb several times a day.
- Severe cases may improve with high-dose oxytetracycline administered slow IV (44 mg/kg diluted in 1 litre of sterile saline) (Lokai et al. 1985).
- If secondary to lameness, provide analgesia and treat the inciting cause.
- Lower the heels. (Elevating the heel provides temporary comfort, but will not correct the problem in young foals.)
- Protect the toe or dorsal portion of the lower limb from excessive wear.
- Splinting and casting should be used with caution.
- Consider euthanasia for severe cases that don't show improvement.



Figure 18.1.2 Bilateral carpal flexural deformity preventing full extension of both limbs.

Flexor tendon laxity

Foals, particularly when premature, can be born with very weak tendons.

Clinical signs

Flexor tendons are weak so the foal stands on the plantar/palmar aspect of the pastern/fetlock. This condition is more common in the hindlimbs.

Treatment

Rest in a small enclosure. Ensure the foal is stable enough to be able to suckle, or get the owner to support the foal for suckling every hour. Splinting or casting the legs is not recommended as, in the long term, it will make the tendons weaker and could result in permanent damage. Very light bandages, which do not provide support, can be applied to protect the legs from injury if the foal is weight bearing on the plantar/palmar skin of the fetlock/pastern.

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