# Neonatal Diseases in Llamas and Alpacas

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Most people working with camelids are familiar with the emergency of the crashing cria. Given the inevitable emotions involved on the part of the owner that doesn't have a large herd, it is important to maintain a clear head when evaluating these cases and to be thorough while acting promptly to deal with life-threatening conditions. As detailed elsewhere in this publication, close monitoring of neonates is advisable such that any deviation from normality can be detected early and the potentially fatal consequences of neonatal illness avoided. Neonatal body systems are not as resilient as those of adult animals and relatively minor events may have quite dramatic adverse effects on the cria. They are also much more vulnerable to exposure to pathogens. Thus, it is not uncommon to be presented with a cria that appears to be completely unresponsive and may have major biochemical or hematological derangements. However, crias do seem to be remarkably responsive to intensive care in contrast to foals, and those that respond well in the first 24 hours of treatment will often proceed to make a full recovery. The exceptions are those crias having congenital defects that will not be amenable to treatment, such as choanal atresia, or major cardiac abnormalities; it is especially important to evaluate crias fully on presentation for any defects that may potentially make costly treatment failure inevitable and cause further distress to the cria, its dam, and the owner.

The most common problems in neonatal crias (less than 2 weeks of age) are problems relating to environmental conditions (hypo- or hyperthermia), nutritional management (ie, failure to nurse resulting in dehydration, hypoglycemia, or failure of passive transfer [FPT] and subsequent sepsis), prematurity, and congenital conditions. Often there will be a combination of these factors. Crias born following a difficult birthing or C-section delivery are also more susceptible to development of hypoglycemia and FPT caused by fetal and maternal exhaustion and delayed nursing. They may also have cerebral hypoxia as a result of prolonged parturition and need additional attention in ensuring adequate intake of nutrition for the first few days of life.

The clinical presentation and management of neonatal disease in crias is not unlike that in foals. Intensive management is frequently required, so referral to a hospital

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facility that is able to offer 24-hour care and in-house diagnostics should be carefully considered and offered to the owner.

## EMERGENCY MANAGEMENT OF NEONATAL CAMELIDS
### Assessment

A complete history should be obtained from the owner and a thorough physical examination should be performed. The normal body temperature for crias is 37.8 to 38.9°C(100–102°F) while normal heart rates vary from 70 to 100 beats per minute and respiratory rates from 20 to 30 breaths per minute. It is important that the initial physical assessment is done as quickly as possible in order to assess the need for immediate intervention and stabilization measures, such as oxygen therapy or fluid therapy, to address severe hypoglycemia. Look for any congenital defects, such as choanal atresia, cleft palate, or umbilical hernia, and ensure that the urinary and anal openings are patent. Make sure that there are no signs of an infected umbilicus. Check the heart carefully for murmurs but remember that hypovolemia, sepsis, and electrolyte abnormalities can be responsible for murmurs, but normally only up to grade III/VI. Pay particular attention to indications of sepsis, such as hyperemia of the mucous membranes and hypopyon (pus in the anterior chamber of the eye). If hypopyon is not detected and treated with mydriatics, such as atropine drops, formation of fibrin strands may occur within the anterior chamber resulting in development of synechiae; these may subsequently result in glaucoma and necessitate enucleation. Look for evidence of prematurity and listen carefully to the lungs for any indication of premature-lung syndrome.

Arterial blood-gas analysis can be very useful in evaluating the sick neonate. One of the easiest sites to collect this sample from is the saphenous artery as it passes over the inside of the hind limb just caudal to the stifle. A 25-g needle is recommended for this technique to minimize the extent of any hematoma.

### Placement of an Intravenous Catheter

By the time most crias present for evaluation, they are often going to require intravenous fluid therapy of some sort. Therefore, it is reasonable to place an IV catheter early in the evaluation so that emergency treatment can be initiated while the examination is under way. Furthermore, blood samples can be collected from the catheter immediately after sterile placement for hematology, biochemistry, and culture, thereby reducing the number of venipuncture sites. Hematoma formation is even more common in crias than in adults and each venipuncture increases the chance of introducing pathogenic bacteria into the bloodstream of the neonate. Rapid evaluation of glucose on a handheld glucometer and packed cell volume (PCV) and total protein assessment can give useful early clues for therapy. If an in-house hematology analyzer is not available, then a fresh blood smear should be examined to guide diagnosis and antibiotic choice. Arterial or venous blood-gas analysis is also ideal. There are some good quality handheld blood-gas analyzers available now, but their accuracy in camelids is so far untested. They may prove to be an excellent tool for practitioners working with camelids in the field because of the rapidity with which results are available. This may greatly enhance diagnostic ability and aid in decision making on the farm.

For crias, an 18-guage, 2-inch Angiocath catheter is ideally placed into the jugular vein for up to 5 days and is easy to place. For crias that may require a catheter for longer periods, then a 16–guage, polyurethane over-the-wire catheter (MILA International, KY) would be more appropriate because they are less thrombogenic and they become dislodged less frequently. The latter are a little more technically demanding to place and require strict attention to sterility during placement. The jugular vein in crias

is easier to catheterize than those of adults because the overlying skin is not as thick and the vein can be readily visualized once the skin has been clipped. Peripheral veins, such as the cephalic vein, are hard to visualize in crias and are difficult to maintain because they are highly positional. They are also not suitable for plasma administration. It is important that the handler has proper control of the cria to minimize vascular trauma during placement. Local anesthetic infiltration of the site of venipuncture greatly enhances catheter placement (0.2–0.3 mL lidocaine). The author prefers the cria to be positioned in sternal recumbency with the head held so that the neck is straight and immobilized; others prefer the cria to be positioned in lateral recumbency. The drawings in Fig. 1 illustrate the author's preferred technique. In crias with hypovolemia, the blood pressure may be so poor that it is not able to displace a column of heparinized saline within the catheter. Therefore, in crias it may be advisable to flush the catheter and then let the flush drain through prior to placement.

If venous access is not possible because of hypovolemia, intraosseous infusion may be achieved by placement of an 18-guage spinal needle into the proximal femur. Fluids may be administered just as easily by this route and will soon restore blood pressure so that an intravenous catheter may be placed.

### Emergency Therapy

The typical crashing cria is likely to be hypoxic and should have oxygen administered until the results of blood-gas analysis are available. Oxygen can be administered by way of a face mask or nasal insufflation tubes; those used for humans work well for crias and they can be secured around the back of the head. They are generally better tolerated than face masks and free up the operator to do other tasks as it is not necessary for them to be constantly present holding a mask in place. If these are not available, a small-diameter flexible catheter can be inserted up one nostril to the level of the medial canthus and secured in position for oxygen supplementation. If intubation is required for a cria that is not breathing by itself, an endotracheal tube (size 4–6) can be placed with the aid of a long-bladed laryngoscope and a dog urinary catheter can be used as a stylet to guide placement.

Once the IV catheter has been placed, the blood-glucose concentration should be checked on a handheld glucometer and any hypoglycemia should be addressed. If hypoglycemia is severe, it is recommended to give a slow infusion of about 50 mL of dextrose-containing fluid (ideally about 10% dextrose) over about 5 to 10 minutes. This provides a safe volume of resuscitation fluid and this can be repeated 20 to 30 minutes later if required. Large volumes of fluid can easily induce pulmonary edema in crias. Boluses of hypertonic dextrose solutions (eg, 50% dextrose) may worsen any preexisting CNS problems and should generally be avoided.[1] Further fluid-therapy decisions can be made once the results of biochemistry are available. In general, maintenance fluid-therapy requirements are about 50% higher than those of adults. The author uses a guide of 1.5 mL/lb/hr for maintenance in crias (or 3.3 mL/kg/hr) and this can be administered by continuous rate infusion using an infusion pump. When crias are more active and able to get up and nurse for themselves, bolus administration of fluids every 2 to 4 hours can be easier to manage since the cria does not need to be separated from its dam. When administering glucose or dextrose to sick crias, it is important to monitor blood-glucose concentrations serially as crias do appear to be prone to a hyperglycemic hyperosmolar syndrome (discussed elsewhere in the issue).[2] For this reason, checking blood-glucose concentration is warranted from the perspective of assessing and managing hyperglycemic crias.

A plasma transfusion may be warranted in certain situations. In an emergency situation with a neonate less than 7 days of age, provided congenital abnormalities have
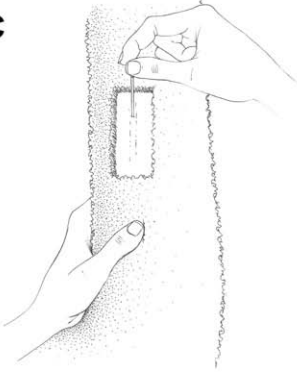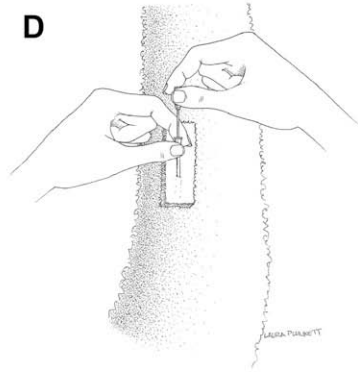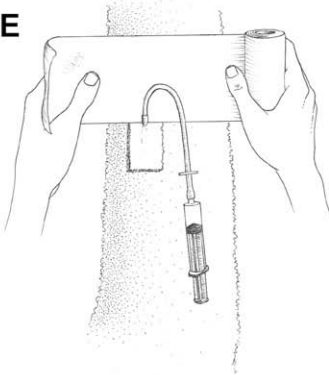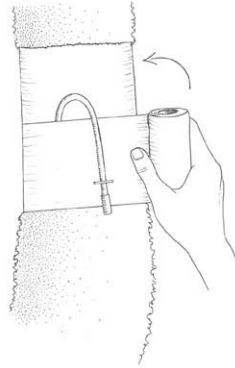
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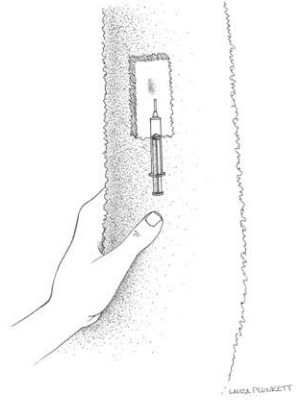
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been ruled out, plasma is almost certainly going to be required since the cria is likely to have some degree of FPT, regardless of the underlying cause. As detailed elsewhere in this issue, there are several ways to assess adequacy of passive transfer. Since the most accurate radial immunodiffusion (RID) test takes 24 hours to run, a more immediate, but crude, assessment can be made using a refractometer reading of total solids as long as the clinician is aware of the limitations of this method. If the refractometer reading is really low (<4.5 g/dL), this is consistent with FPT; if the cria is dehydrated, the actual reading would be even lower. Above this it may be difficult to be sure, so protein and globulin concentration determination on biochemistry results may be more useful. However, Dolente and colleagues[3] found that there was no significant difference between the total protein concentrations in septic crias with positive blood cultures regardless of whether the cria was shown to have FPT or not. The authors suggested that there may be many factors relating to sepsis that make total-protein assessment an unreliable indicator of IgG concentration.

Plasma should be administered at 20 to 25 mL/kg, but higher doses than this are regularly given owing to the average size of plasma bags available commercially (about 300 mL). Plasma is much more effective than crystalloid fluids at expanding volume in very ill neonates, since these crias often have increased endothelial permeability caused by inflammatory changes as a result of sepsis and endotoxemia. Clinically, the observed response during a plasma transfusion can be quite rewarding. The safest method of plasma administration is intravenously using an administration set with an in-line filter that filters out any protein clots. The author typically gives 1 mg/kg of flunixin meglumine intravenously as a premedication about 5 to 15 minutes before commencing the infusion. Very rarely, anaphylactic reactions may occur, therefore it is important to start the transfusion slowly in order to minimize the potential effects should anaphylaxis occur. Normally, it is advisable to give the transfusion over 30 to 40 minutes while monitoring the heart for excessive tachycardia or rhythm

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**Fig. 1.** (*A*) Cria restraint. The cria is placed cushed on a table with the handler drawing the cria to the edge of the table against him/herself for added control. The right hand is placed over the shoulders to prevent the cria from jumping up and the left hand cradles the cria's jaw ensuring that the cria can breathe easily. It helps to hold the cria's head against the chest to give added control. It is important that the cria's neck is in a straight line with its body and that the head is not pulled across to one side, which is less comfortable for the cria and makes it harder for catheter placement. (*B*) Local anesthetic placement. Following clipping of a rectangle of hair from over the jugular vein on the right side of the neck, the site is prepared aseptically. A subcutaneous bleb of local anesthetic (about 0.2–0.3 mL lidocaine in a cria) is placed over the jugular vein using a 25 g needle. (*C, D*) Catheter placement. The catheter is placed through the site of local anesthetic infiltration directly into the jugular vein. Once the catheter is in the vein, advance it just slightly so that it is properly seated in the vein. For a right-handed person, the stylet is held between thumb and finger using the right hand and the catheter is advanced over the stylet into the vein using the left finger and thumb placed on the hub of the catheter. It helps to anchor the right hand fingers against the cria's neck in order to help prevent the skin from rucking up as you slide the catheter into the vein. Blood can be collected at this point for diagnostics. (*E, F*) Attach extension tubing and secure in place. Extension tubing containing heparinized- flush solution is attached to the hub of the catheter. Use the shorter extension sets that have a clamp or valve. It is not necessary to suture the catheter in place; an elasticon bandage wrapped first behind the catheter and over the hub in order to secure it in place works well. Over-the-wire catheters should be sutured. Place a sterile injection port on the end of the extension tubing; this should be changed every 24 hours.

disturbances and respiratory rate, but larger volumes (40 mL/kg) should be given over an hour. Anecdotally, the author has known 20 mL/kg to be given over 5-10 minutes due to operating within a veterinary teaching hospital without adverse consequences although this rapid an infusion is not recommended!

Intraperitoneal transfusion (high right flank) has been done regularly by practitioners, but this approach is not recommended and has the following disadvantages: if given rapidly (5–10 minutes) it results in abdominal pain caused by the large volume given relative to peritoneal cavity space; peritonitis may result if adequate aseptic technique is not used or nonsterile plasma is given; adhesions may result from fibrin deposition and peritonitis and this may result in colic up to 2 years later; inadvertent puncture of abdominal organs may occur resulting in hemorrhage or punctured bowel. Furthermore, intraperitoneal transfusions are contraindicated if the cria is already showing signs of sepsis and these may be as vague as a failure to gain weight (or weight loss), or failure to nurse.

## NEONATAL DISEASE CONDITIONS
### Hypothermia and Hypoglycemia

Newborns are especially susceptible to hypothermia and hypoglycemia. These conditions will often occur together as hypothermic neonates will not stand up to nurse resulting in hypoglycemia. At birth, neonates are subjected to a rapid drop in external temperature and are highly dependent upon nonshivering thermogenesis (NST) to create heat.[4] This process takes place in the mitochondria of brown-adipose tissue and results in an uncoupling of fatty acid oxidation such that heat energy is produced instead of ATP. It is highly dependent on adequate oxygenation, therefore, hypoxemic neonates are more susceptible to the development of hypothermia. Additionally, premature neonates are likely to have inadequate reserves of brown-adipose tissue which develops in the fetus under the influence of NST inhibitors, leaving them at increased risk of hypothermia at birth. Shivering may provide an additional source of heat but the likely impact is small in neonates becase of their small size and immature musculature. The presence of brown-adipose tissue has not been specifically documented in camelids to date, so it is uncertain to what extent NST plays a role in averting hypothermia in crias. Thyroid hormones are thought to play a role in enhancing thermogenesis in the newborn: neonatal llama crias have been shown to have very high thyroxine concentrations at birth, which decreased gradually over the first 90 days of life.[5]

Hypothermic neonates will be depressed and lethargic, will have reduced reflexes and poor ventilation. This, together with poor cardiac function, results in hypoxia acidemia and cardiac dysrhythmias.[6] Hypothermic neonates are susceptible to FPT because they will have a reduced amount of time in which to acquire colostral immunity before gut closure commences.

Crias with hypothermia should be warmed. This will involve placing them in a warmer environment away from drafts, drying them off, if wet, and using external heat sources, such as heat lamps, warmed towels and blankets, hot water bottles, and so forth. A Bair-hugger is an excellent tool for short-term warming available in some hospitals. If heating pads or lamps are used, appropriate insulation from direct heat should be used and care must be taken to ensure that a recumbent cria is turned frequently to avoid overheating. Excessive heat may result in cutaneous vasodilation, which may be detrimental to the cria as it will further reduce core temperature and result in further cardiovascular compromise. The same is true for warm-water bathing. Caution is advised when using heat lamps since some bulbs can present a fire hazard, therefore

they must be positioned above the height of any potential kicks from the dam. Hypo-glycemia may be treated as described above.

### Cerebral Hypoxia

Some crias born following an in utero-hypoxic event, or a prolonged or difficult birth-ing, exhibit signs of cerebral hypoxia (dummy cria syndrome). These crias may be weak or unable to stand and have difficulty nursing if they are capable of trying. They are prime candidates for FPT, malnutrition, and dehydration unless assistance is given. Generally this involves a good level of nursing care with possible IV-fluid support, if necessary. Ensuring adequate nutritional intake and avoidance of FPT are the main goals. Usually after a few days they will tend to get going for themselves.

### Sepsis

Sepsis is defined as systemic inflammatory response syndrome with either docu-mented or suspected infection with bacterial, viral, fungal, or rickettsial agents.[3] It is characterized by pyrexia or hypothermia, tachycardia, tachypnea and either leukocy-tosis or leukopenia. Both gram-negative and gram-positive bacterial sepsis have been reported in neonatal camelids.[3,7] In one retrospective study of crias with culture-posi-tive sepsis, the most common isolates were *E coli, Enterococcus* spp., *Listeria mono-cytogenes,* and *Citrobacter* with gram-negative organisms making up 54% of diagnoses.[3] In these septic crias, hypothermia, tachypnea and tachycardia were common clinical findings and the median age at presentation was 2 days. Pyrexia was not a common finding.

Sepsis is a common sequel to FPT in crias and in neonates of other species. There-fore it commonly presents in crias less than 7 days of age. FPT was a common finding in septic crias in the study previously mentioned[3] and highlights the importance of ensuring adequate colostrum ingestion in newborn crias. Presenting complaints in crias with sepsis include lethargy, depression, failure to nurse or even a failure to gain weight or a loss of weight over a 24-hour period. As previously mentioned, hypo-thermia, tachycardia, and tachypnea are relatively common clinical findings. Addition-ally, there may be evidence of dehydration and injected mucous membranes. From personal experience, crias presenting with a history of failure to gain weight or a loss of weight over the preceding 24 hours may have normal physical examination findings, but hematology performed at this time may reveal a significant leukocytosis or leukopenia with a left shift supportive of the onset of sepsis. Blood work may also reveal findings suggestive of FPT, such as hypoproteinemia and hypoglobulinemia. Treatment at this stage may often avert a crisis presentation 12 to 24 hours later and supports the recommendation of being proactive in terms of performing diagnos-tics in neonatal camelids. Crias will deteriorate rapidly if early signs of sepsis are not recognized and treated. Additional laboratory findings later on in sepsis may include hypo- or hyperglycemia, azotemia as a result of dehydration or kidney failure, and electrolyte and acid-base abnormalities. Eventually other body systems may start to show evidence of dysfunction as the consequences of sepsis become more widespread.

Dolente and colleagues reported that leukopenia and neutropenia were more common in gram-negative infections in crias while gram-positive infections (eg those caused by *Listeria monocytogenes*) were more commonly associated with leukocy-tosis and neutrophilia.[3] This knowledge may be useful in guiding initial antibiotic therapy since blood culture results are unlikely to be available for at least 48 hours after admission. For a cria showing leukopenia, an intravenously administered antibiotic regimen that has a good spectrum of activity against gram-negative organisms is likely

to be a good choice. In the author's experience, combination therapy of a penicillin with an aminoglycoside, such as gentamicin or amikacin results in the best clinical response in these cases. The pharmacokinetics of gentamicin have been investigated in healthy adult llamas.[8,9] Gentamicin was shown to have a prolonged half-life in llamas compared with other species and a dose of 4 mg/kg was shown to reach desired peak serum concentrations.[8] These investigators recommended increased dosing intervals in llamas. Another study found no adverse effects after a single dose of 5 mg/kg given intravenously.[9] The author has frequently used gentamicin at a dose of 5 mg/kg q24 IV for 5 days in adult and neonatal camelids without complications when given in conjunction with intravenous- fluid therapy.

Intravenous-fluid therapy is a vital component of therapy in septic crias to provide cardiovascular support as well as replacement and maintenance fluid and energy requirements. Plasma transfusions are usually required to treat any underlying FPT, but plasma is also very effective at expanding volume in septic crias as they will often have increased endothelial permeability caused by inflammatory changes as a result of sepsis and endotoxemia. Crias can develop hyperglycemia, hypernatremia, and hyperosmolarity in response to dehydration and stress.[2] Sepsis can exacerbate the syndrome. Camelids are prone to hyperglycemia as a result of stress as they are known to exhibit poor pancreatic responses resulting in slower glucose clearance than other species.[10] Crias appear to be able to clear glucose more rapidly because of stronger insulin responses and they are also better able to respond to exogenous insulin, but this may not be true in sick crias.[11] Hypernatremia results from loss of water as a result of glucose diuresis. Even on isotonic fluids, such as lactated Ringer's solution, crias have a tendency to become hypernatremic, therefore the author prefers to use fluids with lower sodium concentrations (eg 0.45% sodium chloride with 2.5% dextrose as long as there is no preexisting hyperglycemia). It is important to monitor glucose concentrations and electrolytes when managing sick crias. Insulin therapy (at a dose of 0.2 IU/kg SQ[11]) is indicated in hyperglycemic crias to prevent ongoing glucose diuresis, and supplementation of fluids with potassium chloride is often required to prevent or treat hypokalemia.

Further management of septic crias involves regulation of body temperature, provision of oxygen in critical cases, and monitoring of hydration status and hematological responses. Crias must be monitored for development of hypopyon, uveitis and conjunctivitis. If hypopyon is observed, administration of mydriatics, such as atropine drops, can minimize the risk of synechiae formation which may subsequently result in glaucoma and necessitate enucleation. Moribund crias are at risk of corneal ulcers because of reduced blinking reflexes. Twice-daily weighing provides a useful guide to progress. Fluid therapy can be gradually tapered off once biochemical abnormalities have been corrected and the cria is able to nurse.

Finally, it is important to check whether or not the dam has sufficient milk and if supplementation is required. Nutritional management is just as important as all the other considerations in managing sick crias. If the dam has milk, the cria should preferably be assisted to nurse or the dam milked out to bottle feed the cria. Alternatively, the cria can be supplemented using warmed goats' or cows' milk. Llama milk has been shown to have higher sugar content (6.5%) and less fat (2.7%) than the milk of other domestic ruminants and it appears that either goat or cow milk is the most suitable alternative.[12]

### Meningitis

Meningitis is a potential sequel to neonatal septicemia but fortunately occurs rarely. Cases of meningitis or meningoencephalitis due to *Listeria monocytogenes*,[13,14]

*E coli*,[13,15] *Salmonella newport*[16] and *Streptococcus bovis*[17] have been reported in crias.

Crias with meningitis or meningoencephalitis may show remarkably variable and vague clinical signs. These may include weakness, depression, inability to stand or elevate the head, tremors, ataxia, opisthotonus, and seizures. In one case seen by the author, the only clinical sign on presentation was abdominal discomfort and this progressed to signs of CNS depression within several days. Another case presented because it was spending increasing periods of time sleeping; meningitis with secondary hydrocephalus was subsequently diagnosed based on cerebrospinal fluid analysis and computed tomographical evaluation of the brain. Blood work may be unremarkable unless sepsis has also developed, but low-globulin concentrations may suggest FPT. These are cases to watch closely for development of septicemia or meningitis and to treat appropriately for FPT.

There is a reasonably good chance of a CSF tap being useful in terms of exhibiting leukocytosis on cytology, especially with suppurative inflammation (Whitehead, Anderson, Saville, unpublished observations, 2005). One report in the literature describes septicemia with meningoencephalitis in a neonatal cria in which the CSF tap showed suppurative inflammation.[13] A 3.5 month-old llama cria with suppurative meningoencephalitis had a massive white-cell count on CSF (10,886 cells/$\mu$L) from which a pure growth of *Listeria monocytogenes* was cultured.[14] Other cases of meningitis and encephalitis in neonatal crias have been reported but CSF was not often evaluated.[15,16] None of the animals reported in the literature survived. This fact and clinical experience of similar cases suggest that crias with meningitis or meningoencephalitis have a guarded prognosis even with aggressive treatment.

### Vertebral and Brain Abscessation

Vertebral or brain abscessation may be another unfortunate sequel to sepsis in crias. Brain abscessation was reported in a 2-week-old alpaca cria caused by *E coli*[15] and in a 1-month-old alpaca cria from which *Fusiformes* spp were cultured.[18] The first alpaca died, whereas the second alpaca made a successful recovery following surgical removal of the abscesses by way of craniotomy. The author is aware of at least two cases of brain abscessation that were successfully treated with medical therapy (Claire Whitehead, unpublished observations, 2005).

Vertebral abscessation has been diagnosed by the author in several crias under 6 months old presenting with paresis or paralysis of the hind limbs in which vertebral abscesses were found in the thoracolumbar region. Clinical signs reflect the site of abscessation and the extent of neurological dysfunction caused by compressive and local inflammatory effects.

Discospondylitis has also been reported as causing neurologic signs in a 2-month-old llama, with the onset of ataxia as early as 1 week of age, progressing to tetraparesis.[19] A lesion was suspected between C3 and C4 on the basis of sclerosis of the vertebral end plates seen on plain cervical radiographs. Treatment was attempted with florfenicol and antiinflammatories, and later ceftiofur, but this was unsuccessful and the cria died.

### Umbilical Infections

Umbilical infections and abscesses do occur in camelids,[20,21] although in the author's experience they are relatively uncommon in comparison with calves. This difference in apparent prevalence may be caused by current comparative differences in husbandry and the cleanliness of the birthing environment. The infections are most likely caused

by contamination of the umbilical stump in the presence of some degree of failure of passive transfer of immunity.

Infections of the umbilicus may result in heat, pain, and swelling of the umbilicus. Deeper infections of the umbilical arteries, veins, urachus, or peritoneum may result in more severe clinical signs, such as lethargy, anorexia, depression, and colic depending on the manifestation. Umbilical abscessation may result in loss of weight or condition caused by the chronicity of the infection and may eventually even cause intestinal obstruction, if large enough.[20] Ultrasonographic evaluation of the abdomen is always indicated to investigate a cria displaying signs of abdominal pain or that has swelling of the umbilicus on clinical examination.

Localized umbilical infections may be treated medically with antibiotics and antiinflammatories, while addressing any potential underlying factors such as FPT. Surgical resection of abscesses is recommended by way of celiotomy without entering the abscess cavity.[20] For large abscesses, if surgical resection is not possible or feasible, the abscess may be drained and marsupialized by suturing the capsule to the abdominal wall for subsequent lavage and drainage.[20]

Crias with evidence of umbilical infection should be evaluated for concurrent infections, such as pneumonia or septic arthritis, because of the possibility of bacteremic spread.

### Septic Arthritis

This is another potential sequel to FPT in crias. Fortunately, it does not appear to be particularly common since the prognosis is poor, especially where there is multiple joint involvement. Affected crias will present with lameness and they may be non-weight bearing. Joints will feel hot, swollen, and painful to the touch. Radiographs may support the diagnosis and highlight any bone involvement while arthrocentesis can be used to confirm the diagnosis by cytology, evaluation of fluid total protein, and culture. Aggressive therapy with systemic and intra-articular antibiotics is required and joint lavage should also be performed, and repeated daily as required.

### Neonatal Diarrhea

Diarrhea is an important disease in neonatal llamas and alpacas. One study found that diarrhea was the most common cause of morbidity in the preweaning period, affecting some 23% of crias (Sharpe et al, unpublished data, 2000). This study monitored 250 crias on four farms over a 5-year period. Commercialization of alpaca breeding in the developed world especially in the United States, Australia and Europe, for fiber production has led to increased stocking densities inevitably exposing young stock to higher concentrations of potential pathogens. Nutritional factors, such as over-feeding with bottle-fed orphans, may play a role but infectious pathogens are an increasingly important factor in neonatal diarrhea. A knowledge of the types of agents involved and at what age these infectious agents are likely to cause disease is important in deciding which diagnostic tests to perform and in initiating treatment. Additionally, the clinician must realise that failure to reach a diagnosis and treat the cause of diarrhea effectively will often lead to chronic diarrhea, which may ultimately result in chronic renal failure. Failure of passive transfer predisposes the neonate to bacterial, viral and protozoal agents but each tends to have a characteristic clinical presentation and this may help guide the clinician with regard to therapeutic options.

The main infectious causes of diarrhea in llama and alpaca crias are listed in **Table 1** including the ages at which they are likely to be diagnosed and the diagnostic tools available. Nutritional causes, antibiotic-induced diarrhea and metabolic disturbances,

**Table 1**
**Main infectious causes of diarrhea in young alpacas and llamas in the United States**

| Pathogen | Age of Cria Affected | Diagnostic Testing |
|---|---|---|
| *E. coli* | <7 days | — |
| Coronavirus | From 10 days | Electron microscopy of feces |
| *Cryptosporidium* | From 7 days | Fecal analysis, acid-fast staining of fecal smear, ELISA, PCR to speciate & identify source |
| *Giardia* | From 7 days | Fecal analysis, ELISA |
| *Coccidia* | From 21 days | Fecal analysis |
| *Salmonella* | Rare, any age | Fecal culture |

such as portosystemic shunts should also be considered as differentials. Nematodiasis may cause diarrhea in crias older than 2 months.

### E. coli Diarrhea

*E. coli* diarrhea often occurs in combination with neonatal septicemia. Affected neonates, typically 3 to 7 days old, are severely ill with profuse, watery diarrhea, lethargy, dehydration, and may have abdominal distension. Colibacillosis occurs secondary to FPT in neonates, but can occur secondary to other gastrointestinal diseases, such as viral enteritis in slightly older animals. If *E. coli* septicemia and diarrhea are not treated intensively early on, rapid progression of clinical signs may occur. Water and electrolyte losses can be severe, with bicarbonate loss being particularly significant.

The enterotoxigenic *E. coli* are a leading cause of neonatal loss in livestock but have not been specifically identified in llamas and alpacas. Likewise, enteropathogenic *E. coli* have not been reported in llamas or alpacas.

Gram-negative bacterial infections were reported in five neonatal llamas and one alpaca.[7] All were aged 1 to 5 days and had positive blood cultures. Three of the crias presented with diarrhea and all three died soon after admission to the hospital. *E. coli* was cultured from 3 of the 6 cases, though it is not clear whether these were the crias that presented with diarrhea. However, all three of the crias that had diarrhea were necropsied and had lesions of hemorrhagic enteritis evident grossly. Bacterial culture did not yield any *Clostridium* sp in these cases; assays for clostridial toxins were not performed. Diagnostic tests for viral pathogens or parasites were not done.

The author's experience with suspected clinical cases of *E. coli* diarrhea is that affected crias are often leukopenic with a degenerative left shift neutrophilia. When a cria aged less than 7 days presents acutely ill with diarrhea and has these characteristic findings on complete blood count, broad-spectrum antibiotic administration is indicated until blood-culture results are available and should include gram-negative coverage. Supportive therapy, including intravenous-fluid therapy, is also likely to be required.

### Viral Diarrhea

Both rotavirus and coronavirus have been identified as causing diarrhea in neonatal llamas and alpacas. Coronavirus was the most common pathogen causing diarrhea in unweaned crias in one recent study.[22] It was identified by electron microscopy in 42% of cases and affected 64% of herds studied with an age distribution from 10 to 150 days at the time of diagnosis. In the same study, this was the only pathogen

involved in outbreaks that also affected adults. Coronavirus also was found to cause diarrhea at any time of year in contrast to coccidiosis, in which case outbreaks coincided with wetter times of year (Fall and Spring). In contrast, rotavirus was found in only 2% of cases.

Parreño and colleagues investigated the presence of rotavirus and coronavirus in two farms of captured guanacos in Patagonia (Argentina).[23] Both farms suffered severe diarrhea outbreaks affecting 100% of the herds, and fatal in 83% of cases. Animals were aged between 1 day and 4 months at time of capture, kept in small yards, and raised on powdered cow's milk. In all cases disease was acute in onset and the stool was dark-green. Dehydration occurred and death followed within 2 to 6 days. Affected animals were 7 to 40 days old. Rotavirus was detected in the feces from two neonates (aged 2 and 7 days). Molecular characterization of rotavirus isolates from newborn guanacos in Argentina were shown to be closely related to other bovine rotavirus strains from other parts of the world.[24]

Puntel and colleagues conducted a serological survey among 390 llamas in Argentina testing for antibodies to a variety of viruses including bovine rotavirus.[25] They found antibody prevalence for rotavirus of 88% with positive animals from all nine of the farms tested. The authors surmised that these findings suggest a high susceptibility of llamas to bovine rotavirus. No clinical cases of diarrhea were reported on the farms of study. Mattson reported detection of coronavirus by electron microscopy in two young llamas with diarrhea.[26] One of these cases was aged 12 days and the other 9 months.

Treatment of viral diarrhea is mainly supportive. It is important to rule out and if necessary treat for other pathogens which may be involved concurrently. Various monoclonal antibody vaccines against viral pathogens are available for oral administration to calves and lambs. These may be used safely in camelids on farms experiencing outbreaks of viral diarrhea but they are of unknown efficacy in these species.

### Cryptosporidiosis

*Cryptosporidium* species are zoonotic protozoan pathogens and can cause severe and sometimes fatal outbreaks of diarrhea in neonatal animals and immunocompromised individuals. *Cryptosporidium* species lack host specificity. Numerous species have been recognized, including species affecting birds and reptiles, but *Cryptosporidium parvum* has been investigated most extensively and is believed to be responsible for most mammalian disease.[27] One study investigated the pathogens involved in diarrhea affecting 45 unweaned llama and alpaca crias and *Cryptosporidium* spp were isolated in 9% of cases.[22] *Cryptosporidium* may be a secondary pathogen but one recent study found no other gastrointestinal tract pathogens in 13 out of 20 alpaca crias diagnosed with cryptosporidiosis, suggesting that it may occur as a primary pathogen.[28] Cases of cryptosporidiosis reported in llamas and alpacas have been in crias aged from 7 days to 6 months.[28–30] The fecal material at presentation varied in consistency from watery to pasty, and in color from white to tan or green, if crias were old enough to be grazing.

Infection occurs by fecal-oral route from ingestion of contaminated food or water. *Cryptosporidium* oocysts are extremely resistant to decay in the environment and to most disinfectants. As few as 10 oocysts can induce disease in susceptible individuals, while clinically affected calves can shed massive numbers of oocysts during infections forming a significant source of infection for other animals.[27] Additionally, asymptomatic adult cattle are thought to be a major source of environmental contamination. The exact mechanism by which *Cryptosporidium* attaches and invades

enterocytes is poorly understood at the present time. However, it is known that following attachment, *Cryptosporidium* forms a parasitophorous vacuole within the host cell (intracellular but extracytoplasmic) bounded by a parasitophorous vacuolar membrane derived from the host-cell membrane. This protects the organism from the hostile environment of the gut lumen and the host cell's defenses. An additional feeder organelle membrane in *Cryptosporidium*, as opposed to other coccidian parasites, is thought to be primarily responsible for nutrient uptake from the host cell. Inclusion of *Cryptosporidium* within enterocytes reduces gastrointestinal absorption in affected individuals caused by loss of epithelium, villous atrophy, and crypt hyperplasia leading to dehydration and electrolyte imbalance (malabsorptive diarrhea). Maldigestion also occurs as a result of the loss of membrane-bound digestive enzymes, such that weight loss and emaciation in neonates is typical. Autoinfection can occur. The author has diagnosed renal failure in multiple crias with cryptosporidiosis. The mechanism of kidney damage is presumed to be prolonged states of subclinical dehydration leading to reduced renal perfusion and tubular necrosis.

The simplest method of diagnosing cryptosporidiosis is by examination of fecal smears using modified acid-fast stains.[27,31] Detection of oocysts can be improved by the use of immunofluorescence microscopy and ELISA. Recently, ELISA tests for both *Cryptosporidium* and *Giardia* oocyst antigens have improved sensitivity and can be easily applied in practice situations. PCR allows speciation and the possibility of identifying the potential source of infection.

Effective specific treatment for cryptosporidiosis remains elusive. The mainstay of therapy involves supportive care with intravenous fluids or total parenteral nutrition caused by malabsorption and maldigestion which occurs in this disease. In one recent case series, the authors reported that 20 alpaca crias with cryptosporidiosis generally exhibited clinical signs for at least 7 days, but that infections were mostly self-limiting, and that good supportive care resulted in a successful outcome in 16 crias.[28] Supportive care included partial parenteral nutrition in 19 of the 20 crias.

Fecal shedding of *Cryptosporidium* oocysts has not been demonstrated in asymptomatic South American camelids. Rulofson and colleagues found no *Cryptosporidium* oocysts in 354 llamas aged between 3 weeks and 23 years.[32] However, six camels at the Barcelona Zoo were all found to shed *Cryptosporidium* oocysts over a 1-year period, mostly because of reinfections, despite being asymptomatic.[33]

### Giardia

*Giardia* infection primarily occurs from contaminated water sources and is a zoonotic disease. The oocysts can survive about 3 months in water at 4°C. Since the organism affects the small intestine where it causes villous atrophy, a malabsorptive diarrhea results in dehydration and weight loss.

*Giardia* was the pathogen responsible for 18% of 45 cases of diarrhea in unweaned llama and alpaca crias[22] and in 33% of 58 crias in another hospital-based retrospective study.[34] *Giardia* was reported for the first time in a llama in 1987.[35] That llama was several months old and asymptomatic at the time of fecal examination. Fecal shedding of *Giardia duodenalis* was reported in 3.4% of 354 asymptomatic llamas with crias being significantly more likely to shed oocysts than older llamas.[32] In that study, risk factors that increased the likelihood of oocyst shedding included having more than 10 yearlings on the property, smaller pen sizes, and large unit sizes of more than 20 animals.

In the authors' experience, oral fenbendazole at 50 mg/kg once daily for 5 consecutive days is effective at treating giardiasis in llamas and alpacas.

### Coccidiosis

Coccidiosis is most commonly diagnosed in neonates and juveniles. Cebra and colleagues isolated *Eimeria* spp out of 13% of 45 unweaned llama or alpaca crias with diarrhea; these six crias were between 21 and 60 days old.[22] Adults are more resistant to clinical disease because of mature immune systems and prior exposure. Coccidiosis is typically associated with overcrowding and poor hygiene. The pathogenesis and severity of the clinical signs observed may be associated with the number of coccidia ingested. Reinfections from a contaminated environment can cause an excessive coccidia burden that may be fatal.[36] Coccidia undergo sexual and asexual reproduction stages within the intestinal epithelia to produce oocysts and these cause direct damage to the epithelial mucosa of the small intestine resulting in enteritis and diarrhea. Tenesmus may be observed. Diarrhea may be hemorrhagic and contain shreds of sloughed mucosa. As a result, nutrient malabsorption and subsequent poor growth ensues. Cheney and Allen report that since the lesions are primarily in the small intestine, fresh blood is rarely seen in affected llamas.[37]

Coccidiosis in camelids is caused by several different species belonging to the genus *Eimeria.* Coccidia tend to be highly species-specific. Six species have been identified that affect South American Camelids and these include: *Eimeria lamae*, *E. alpacae*, *E. macusaniensis*, *E. punoensis*, *E. peruviana* and *E ivitaensis*.[36,38–41] All except the latter of these have been detected in fecal samples from camelids in the United States.[36] A distinguishing feature of the *E. macusaniensis* oocyst is that it is much larger than the other four species, measuring 81 to 107 μm in length, and has a very thick wall.[39] In contrast, the other main four species measure 17 to 40 μm in length and have a thin-walled oocyst.[38]

The life cycle is not well-established for coccidia specific to South American Camelids. However, Foreyt lists prepatent periods for four out of the five species that affect South American Camelids.[42] These vary from as short as 10 days for *E. punoensis* to 33 to 34 days for *E. macusaniensis*. The prepatent periods for *E. alpacae* and *E. punoensis* were established from experimental data using fecal samples from four llamas.[43] The authors have seen clinical coccidiosis in alpacas as early as age 21 days. Such an early appearance of clinical disease suggests that neonates exposed to contaminated environments can become infected in the first few days of life.

Coccidiosis may result in death without necessarily causing premortem diarrhea. Rosadio and Ameghino described an outbreak of coccidiosis on a farm in southern Peru that was suspected to have caused the deaths of 12 alpacas aged between 25 and 35 days. Eight of these cases had been observed with premortem diarrhea, four others had died suddenly. At necropsy, all of the carcasses were observed to be thin and dehydrated. Histopathologically, throughout the mucosa there were large numbers of sexual and asexual stages of a coccidian that was thought to be *E. macusaniensis.* These were causing marked disruption of the epithelial crypt glands. Costarella and Anderson reported a case of ileocecocolic intussusception in a 1-month-old llama cria.[44] A fecal examination had revealed many coccidial oocysts but there had been no signs of diarrhea and a causal relationship was considered but could not be established.

Several other reports have documented the occurrence of coccidial oocysts in feces from healthy South American camelids in the United States.[40,45,46] All studies involved large sample sizes of 239, 144, and 443 animals respectively. In two of the studies, animals less than 1 year old had a higher prevalence of oocyst shedding.[45,46] Method of fecal examination technique can affect the yield of oocysts detected.[46] Flotation solutions with a specific gravity less than or equal to 1.20 (for example,

saturated sodium chloride which is 1.20) may fail to allow detection of *E. macusaniensis* oocysts, which are large and have a high-specific gravity. In comparison, a sugar solution with specific gravity of 1.28 to 1.30 (used at a 1:10 dilution for oocyst detection by centrifugal flotation) gave more reliable results, especially when low concentrations of oocysts were present.

Effective treatment of clinical coccidiosis is achieved using oral sulfadimethoxine (Albon, Pfizer USA) at a dose of 15 mg/kg twice daily for 5 days. Amprolium may also be used orally at 10 mg/kg once daily for 5 days.[47] Amprolium inhibits differentiation of merozoites by acting on first generation schizonts in the small intestine.[48] It does not kill the merozoites: these enter the sexual reproductive phase which ultimately produces oocysts. Therefore, an animal treated with amprolium will still excrete viable oocysts. Clinically affected animals should be isolated and treated. Unaffected animals from the same pen should also be treated since they will have been exposed and may be harboring susceptible coccidian stages.

Good management practices and maintenance of hygienic facilities for young animals should be considered the most important factors in prevention of coccidiosis. Strategic use of anticoccidial drugs may also be considered as a supplemental means of controlling coccidiosis and should be used on a herd basis. Since outbreaks of coccidiosis are common in the wetter months, prophylactic drug regimes should be used during these times of year. Consideration should be given to application of preventative measures before and during stressful events, such as weaning, shearing, or herd movements. Amprolium may be used prophylactically and is added to water at 5 mg/kg body weight daily over a 21-day period. Correct dosage of amprolium is important since it is a thiamine analogue and overdosing may produce clinical polioencephalomalacia. Alternatively, decoquinate may be used more safely when added to feed at 0.5 mg/kg/day for 28 days. Ionophore antibiotics, such as monensin or salinomycin should not be used to prevent coccidiosis in camelids, since they are susceptible to toxicity of these drugs.

### Salmonellosis

*Salmonella* spp do not appear to be a common cause of diarrhea in llamas or alpacas. *Salmonella* was not isolated in any of the 45 cases of cria diarrhea in one study.[22] Seventy-six healthy llamas in California were tested for *Salmonella* shedding and were found to be negative.[32] A field survey included fecal cultures for *Salmonella* in 99 asymptomatic llamas and alpacas residing in Ohio.[49] All 99 cultured negative for *Salmonella*. However, one report exists of septicemic salmonellosis in one 6-year-old female llama and one 6-day-old llama cria (Anderson and colleagues 1995). The cria had *S. typhimurium* isolated from a blood culture. Both cases were fatal but neither displayed any signs of diarrhea.

The authors have cultured *Salmonella* from young (<6 months old) llamas and alpacas with diarrhea. These animals presented with severe, protracted diarrhea and dehydration. Several cases had clinical signs of septicemia (scleral injection, tachycardia, tachypnea, elevated rectal temperature) and were positive on blood culture for *Salmonella* species. A degenerative left shift neutrophilia was present on hematology. The *Salmonella* isolated were of various species (*S. ohio, S. newport, S. choleraesuis*).

### Other Causes

There has been one reported case in the literature of portosystemic shunt in an alpaca cria.[50] This cria had had recurrent episodes of diarrhea up until the time of diagnosis at 5 months of age. The authors postulated that the diarrhea in portosystemic shunt

may be caused by "abnormal hepatic metabolism of nutrients or abnormal bacterial growth in the colon."

Nutritional causes of diarrhea in young llamas and alpacas include sudden access to lush spring pastures, grain overload, or overfeeding milk to bottle-fed crias. The authors have seen crias develop diarrhea when bottle fed with milk replacers or goats' milk at rates greater than 15% of their body weight on a daily basis. Cebra and colleagues reported on six cases of forestomach acidosis including one llama that was 2 months old.[51] The crias from that group were creep-fed a grain mixture of equal parts of rolled barley, rolled oats and cracked corn.

Antibiotic-induced diarrhea should be considered a differential diagnosis in crias with a history of antibiotic treatment, especially if orally administered.

## SUMMARY

This article attempts to cover the most common problems likely to present in neonatal crias and to give guidance on how to approach these cases, particularly how to stabilize the acute presentation of a sick neonate. It is impossible to cover every eventuality and it is important to keep an open mind when evaluating and treating sick neonatal camelids. Rickets is covered elsewhere in this edition. Despite the often moribund presentation of sick neonatal crias, they may be highly rewarding to treat, although hospitalization and 24-hour care is recommended.

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