

Diarrhea in Llama and Alpaca Crias

Christopher Cebra, VMD, MA, MS, DACVIM

College of Veterinary Medicine, Oregon State University, Corvallis, OR 97331

Abstract

Neonatal and juvenile diarrhea are common complaints among owners of cattle, sheep, pigs, horses, and goats. Microbial causes are usually blamed, although in some cases nutritional or other considerations come in to play. The most commonly identified pathogens are viruses and protozoa. These are relatively self-limiting, and clinical signs are more related to fluid and electrolyte loss than anything else. For ruminants and pigs especially, various products have been developed which specifically address water, base and salt loss. Various antibody and vaccine preparations are available to directly combat the causative organisms, but with the exception of *Eimeria*, antimicrobial treatment is usually not considered necessary.

Résumé

La diarrhée néonatale et juvénile est une condition dont se plaint souvent les éleveurs de bovins, de moutons, de porcs, de chevaux et de chèvres. Bien qu'une cause microbienne soit souvent impliquée, la nutrition et d'autres aspects peuvent aussi jouer un rôle. Les organismes pathogènes les plus souvent identifiés sont les virus et les protozoaires. Ces organismes sont rarement hors de contrôle et les signes cliniques sont le plus souvent associés aux pertes de fluides et d'électrolytes. Pour les ruminants et les porcs notamment, plusieurs produits ont été développés afin de contrer la perte d'eau, de base et de sels. Plusieurs préparations d'anticorps et de vaccins sont disponibles pour combattre directement les organismes causant la maladie. Toutefois, les traitements antimicrobiens ne sont pas vraiment nécessaires sauf dans les cas impliquant *Eimeria*.

Introduction

New World camelids are relatively new to North America. Compared to the roughly 103 million cattle, 60 million pigs, 6.9 million sheep and 5.3 million horses in the US, our experiences with the 140 thousand camelids are correspondingly limited. However, camelid owners are very likely to seek veterinary attention when their animals are sick, so we are rapidly gaining experiences.

Older references say little about diarrhea in crias. Bacterial disorders with multisystemic signs, such as coliform sepsis and clostridial enterotoxemia, receive most attention. These are certainly important disorders under certain conditions, but result in clinical pictures very different from neonatal or juvenile diarrhea in other domestic farm animals. Because of the emphasis on bacteria and systemic diseases, treatment recommendations usually emphasize antimicrobial drugs. Without contrary recommendations or data, these practices have continued.

Clinical Syndromes

Diarrhea in domestic farm animals may be roughly divided between sporadic, non-contagious, individual animal problems and contagious herd outbreaks. The distinction may be blurred by management conditions which cause multiple animals to develop non-contagious conditions at a similar time, or by lack of enough like-aged young stock to see an outbreak. The sporadic conditions include sepsis, peritonitis, or non-infectious ulcerative disorders. These may become commonplace in herds with poor hygiene or high prevalence of failure of passive transfer. Contagious conditions include bacteria, viruses and protozoa. Small numbers of susceptible hosts at any one time may disguise the contagious nature of these pathogens.

Historically, the far more common clinical presentation was the affected individual cria. Often, the cria appeared systemically affected and diarrhea was neither profuse nor watery. The diarrhea frequently appeared to have blood in it or was fetid. Other signs included fever, tenesmus, abdominal distention, severe obtundation, recumbency, anorexia, tachycardia, injected mucous membranes, and possibly colic. Clinical pathology evaluation revealed electrolyte or acid-base disturbances that were usually mild and could not account for the degree of obtundation. Abnormalities in the leukogram were often more remarkable: leukopenia or leukocytosis, left-shift, and toxic changes. Thus, the original data about bacterial condition appeared to be justified. Indeed, many crias died despite treatment, and postmortem examination revealed hemorrhage, inflammation and necrosis in the gut wall, occasionally full-thickness perforations of gastrointestinal viscera, and systemic signs of sepsis. Coliform organisms, occa-

sional clostridia and a host of other gut bacteria could be recovered from these lesions.

Occasionally, a cria would present with a more typical diarrhea picture, including profuse, watery diarrhea, dehydration, inappetance and general lack of evidence of systemic disease. Clinical pathology was often similar to that is seen in calves, usually at the milder end of the hyponatremia and acidosis spectrum. In fact, hypernatremia is not uncommon. Efforts to identify pathogens in the feces of those crias were often unsuccessful. *Eimeria*, *Cryptosporidia*, *Giardia* and helminth ova were found occasionally, but with the exception of *Eimeria* in certain herds, repeat isolates from individual herds were rare.

Lack of herd outbreaks may have been due to management as much as anything else. Most camelid farms have a relatively small number of crias born each year, and these may be spaced over several months. Thus, the large numbers of susceptible hosts to multiply the pathogen in the environment may have been lacking in most instances.

Since about 1998, herd outbreaks of profuse, watery diarrhea have become much more common. These were first noted in the Pacific Northwest, but have since been reported in various states. Camelids of all ages are affected, with most outbreaks starting with alpacas coming home from shows, sales, or trips to other premises for reproductive purposes. Llamas may also be affected, but most outbreaks start with affected alpacas. Crias who themselves do not usually go to shows, appear to be more affected by dehydration and electrolyte loss than older animals.

As a last syndrome, soft, non-profuse, unformed feces in crias showing no depression is common. Pathogens are rarely isolated. Poor fecal formation appears to be due to abnormal digestive function, i.e. dietary indiscretion or lack of suitable gut flora. Some people have had success treating these with probiotic agents; almost all resolve in seven to 10 days without treatment.

Establishing the Importance of Individual Pathogens

Several efforts have been undertaken to identify possible causes of diarrhea or presence of possible microbial gut pathogens in crias.

Bacteria

Salmonella - a gram-negative bacterium associated with enterocolitis in many species. Various studies have shown this bacterium to be rare in camelids, although sporadic clinical cases have been reported.

Clostridium - a gram-positive anaerobic bacterium. Anecdotal reports have attributed importance to post-mortem isolates of Type A *Clostridium perfringens* from

the stomach compartments or intestine. These reports are difficult to substantiate, as this organism frequently proliferates postmortem. Other types of *C. perfringens* have been associated with enterotoxemia, but diarrhea in those cases is less common than sudden death or severe systemic and neurologic signs.

Escherichia coli - a gram-negative bacterium commonly isolated from feces of many species. This bacterium has received a lot of attention, but is probably more a cause of neonatal sepsis than any specific GI syndrome. As far as we know, there are not camelid-specific enterotoxigenic strains of *E. coli* like those in calves, piglets and lambs. Coliform organisms and other similar enteric bacteria are important considerations in crias with depression, fever and other systemic signs. If diarrhea is present, it is usually not profuse, and may contain blood.

Protozoa

Eimeria - large coccidian parasites. Camelids in North America appear to be susceptible to four or five species, including the large *Eimeria macusaniensis*. These appear to be species-specific. The prepatent period for *Eimeria* in camelids appears to be about 16 to 21 days. Over that time, there are two rounds of schizogony (asexual reproduction), the first in the lower small intestine and the second in the large intestine. Gametogony (sexual reproduction) then occurs primarily in the large intestine with destruction of the gut epithelial cells. Clinical signs usually appear with gametogony, though diarrhea may precede fecal oocyst shedding by a few days. Because of its predilection for large intestinal damage, *Eimeria* is one of the parasites more commonly associated with diarrhea in camelids.

E. macusaniensis is the largest and slowest-maturing of the coccidia. The prepatent period is over 30 days, and it is common for crias to show clinical signs of disease (weight loss or poor growth, possibly diarrhea) before developing patent infections.

Cryptosporidium - a much smaller coccidian parasite, which can infect many species. In addition to the intestinal epithelium, *Cryptosporidium* has been found in the biliary, renal and respiratory epithelium of some immunocompromised patients of other species. Both *C. parvum* and *C. muris* have been isolated from calves, but no study has ascertained whether both species affect camelids.

Giardia - a flagellate parasite, *Giardia* cysts found in feces of many normal animals, including llamas, thus causing many to doubt its pathogenicity. It appears able to affect many different hosts. Ingested cysts release trophozoites, which attach to small intestinal mucosal cells. Although cell function is impaired, *Giardia* do not reproduce in the cells like *Eimeria*. The number of ingested cysts, and hence number of affected host cells, is

likely to be a predictor of clinical importance of *Giardia* in a specific situation. The incubation period is approximately five days.

Viruses

Rotavirus – a double-stranded RNA virus in the family *Reoviridae*. By electron microscopy, it appears as a thin-rimmed wheel with short spikes. Rotavirus is blamed for diarrheal disease in neonates of most farm animal species, and also occasionally causes disease in adults. It has a fair amount of antigenic variation, and strains have been reported to cross between species. Non-clinical carrier animals or clinically ill ones may introduce the virus to new susceptible hosts. Incubation periods are between about 18 and 96 hours, with primary site of replication being the absorptive epithelial cells of the small intestine. In some cases, large intestinal cells are also affected.

Coronavirus - a large single-stranded RNA virus in the family *Coronaviridae*. Coronavirus is characterized by a spherical virion surrounded by a fringe of radiating peplomers. Coronavirus is blamed for diarrheal disease in neonates of most farm animal species, and also occasionally causes disease in adults. It is genetically relatively conservative, but strains appear to be species-specific. Non-clinical carrier animals or clinically ill ones may introduce the virus to new susceptible hosts. Incubation periods are between about 18 and 96 hours, with the primary site of replication being the absorptive epithelial cells of the small and large intestine.

Other Viruses - A variety of other potential pathogens have been found on different occasions and linked to gastrointestinal disease. These include bovine viral diarrhoea virus (BVDV), parvovirus and others. Evidence for their importance is still lacking.

Diagnosis

Diagnosing the cause of diarrhoea in crias involves getting good samples of abnormal feces early in the course of the disease. Samples must be kept cool and transported to a laboratory quickly, especially for the viruses. If the cria does not defecate readily, a gentle rectal lavage with 10 mL of warmed saline may aid in procuring a sample. If the lavage method is used, cyst and egg counts may be artificially low.

Some pathogens may be detected using light microscopy and standard flotation methods. However, these are the less common pathogens in young crias. Bacterial cultures on feces are also unlikely to be rewarding due to lack of specificity of results. Electron microscopy on fresh fecal samples is very helpful. Delaying microscopic examination or allowing fecal material to freeze or sit too long causes characteristic features of the viral particles to degrade.

Treatment

The degree of electrolyte loss in camelids, even with severe diarrhoea, rarely reaches the severity seen in calves. Thus, electrolyte and acid-base disorders tend to be milder. The predilection for sepsis or enteritis in the past led us to use plasma, antibiotics and non-steroidal anti-inflammatory drugs to treat many crias with diarrhoea. These habits are hard to relinquish and certainly are valid for crias with systemic signs, those with severe depression and mild diarrhoea, and those with clinical pathology or other physical evidence of sepsis.

Unless a specific bacterial or protozoal pathogen is identified, the most important treatments are good supportive care. In the past, that often involved use of oral electrolyte powders developed for calf scours. These powders are high in glucose and salt. Some of our work related to glucose homeostasis in camelids suggests that these powders may contain too much glucose for camelids and result in diuresis of glucose and water. This may further dehydrate the patient, as well as overload it with sodium. Additionally, hypernatremia may be present before treatment, and could be exacerbated by calf electrolyte solutions. Diluting these powders three- or four-fold, using powders or solutions developed for human infants, or supplying enough sodium- and sugar-free fluid in the form of milk or water will help avoid hyperosmolality problems. Some crias will drink electrolyte solutions voluntarily either from a bucket or a bottle. Some require tubing, and the stress of the procedure may outweigh the benefit.

Intravenous fluids are necessary for more severely affected crias. The right jugular vein provides the best access. Unless there is good evidence of severe electrolyte or acid-base disturbances, straight polyvalent, isotonic fluids are best. I prefer to leave the cria on the dam or provide free-choice water and milk replacer at all times to allow the cria to obtain energy and water.

Specific treatments exist for the protozoal pathogens. For coccidia, sulfa antibiotics, amprolium and decoquinate have been used. For *Cryptosporidium*, there is anecdotal information about some agents, but in general, anticoccidials are not known to have a beneficial effect. *Giardia* may respond to high doses of metronidazole or fenbendazole.

Prevention

Hygiene, separation of crias from adults (except their dams), and separation of breeding stock from transient show or breeding animals are very important. These routine biosecurity measures are often ignored due to lack of space on the farm. They were also relatively unnecessary until recently, when contagious outbreaks of disease in camelids became more commonplace.

For protozoal pathogens, pasture rotation and strategic treatment of adults and juveniles may reduce infective oocysts and cysts. For *Giardia* particularly, avoiding damper pastures during wet times of the year may reduce transmission between wildlife hosts and camelids. For coccidian, some farms mill mineral and grain supplements with decoquinatate added.

Colostrum antibody provides some local protection against the enteric viruses, but the concentration of antibody in colostrum drops too quickly to protect the neonate for more than a couple of days. After that period, the neonate's own secretory immunity takes over.

Vaccination of cattle has been shown to increase colostrum antibody, but the duration of protection remains short. Continued colostrum feeding or oral antibody preparations may prolong the protection period, but would have to be continued for several weeks to guide the neonate through the highest-risk period. Vaccination of the neonate is unlikely to provide protection for several weeks and also does not adequately stimulate secretory immunity. Also, vaccines may be less effective against rotavirus because of its greater antigenic variability.