Common field problems in camelids

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Abstract

On the whole, camelids are a hardy group and not prone to diseases. In this session we will discuss common causes of problems in camelids and how to treat and manage these conditions. Some of the topics to be discussed are gastrointestinal parasites, *Mycoplasma haemolamae*, meningeal worm infection, polioencephalomalacia, and more.

Key words: camelids, parasites, *Mycoplasma haemolamae*, meningeal worm infection, anemia

Résumé

Règle générale, les camélidés sont robustes et peu susceptibles aux maladies. Dans cette session, nous allons voir les causes les plus courantes de problèmes chez les camélidés et comment traiter et gérer ces conditions. Parmi les sujets de discussion, il y a les parasites gastro-intestinaux, *Mycoplasma haemolamae*, l'infection par le ver des méninges, la polioencéphalomalacie et plusieurs autres.

Introduction

Although not prone to disease, when taken out of their natural environment, grouped together, and forced to accommodate to what humans think are appropriate surroundings and feed, trouble can arise. As each topic could in itself warrant an hour discussion (or more), this presentation will briefly discuss some of the more commonly encountered problems we see in camelids in the United States.

Common Problems with Camelids

Gastrointestinal parasites can be a problem in both crias and older camelids without appropriate monitoring. Parasites we consider to be most important in older crias through adult camelids are strongyles (including *Nematodirus*), *Trichuris*, *Capillaria*, *Moniezia*, coccidians, and in some parts of the country liver flukes and lungworms. In younger crias, we are concerned about *Cryptosporidium, Giardia*, and coccidians. This will be a brief discussion on adult-type parasites.

There are many different strongyle parasites and in a regular fecal floatation, the many different types cannot be differentiated (with a few exceptions) and are referred to as strongyle-type parasites. The major parasites found in the third compartment (C3 or true stomach) are *Haemonchus, Trichostrongylus, Ostertagia, Camelostrongylus, Teladorsagia*, and *Marshallagia. Haemonchus* is unique as it causes anemia and decreased protein in severe infections, plus the larvae consume blood even before eggs can be detected in the feces. Also, anemia can occur before there is any significant weight loss. To help with diagnosis of *Haemonchus* in a particular animal, Oregon State University and the University of Georgia developed a lectin staining test based on peanut agglutinin that binds specifically to *Haemonchus* eggs and larvae. Small-intestinal worms are *Cooperia, Nematodirus, Trichostrongylus, Lamanema* and *Moniezia.* Compartment 3 and small intestinal parasites rarely cause diarrhea (usually late in severe nfections), but rather weight loss, ill thrift, and low protein.

Trichurus, *Capillaria,* and *Oesophagostomum* are found in the cecum and large intestine. *Trichurus* and *Capillaria* are not commonly found in fecal analysis due in part to adult parasites shedding eggs intermittently, and the eggs do not float very well unless saturated sugar solution and a longer float time is used. Both are clinically important and resistant to treatment. The larvae initially penetrate the small intestine where they mature, and then migrate to the cecum and large intestine and become adults. The adults tunnel into the intestinal wall traumatizing vessels, and in large enough numbers can cause intestinal inflammation and diarrhea.

Camelids are susceptible to several species of liver flukes, with *Fasciola hepatica* and *F. magna* being the most important in the US, particularly in the Pacific Northwest. A wet environment and specific intermediate hosts (snails and slugs) are needed in transmission. These parasites can cause an ill-thrift syndrome characterized by low protein and changes in blood work that indicates liver disease, with specific increases in GGT and bilirubin concentration. Keeping in mind how often camelids travel, any visiting animal showing signs of liver disease should be evaluated for possible liver flukes. Fluke eggs are not routinely found in fecal sugar floatations, so testing is best done by a serum ELISA test for *Fasciola hepatica* at Oregon or Colorado State University. Treatment is with injectable ivermectin-clorsulon combination[a] or with albendazole.[b]

Due to parasites developing resistance to current drugs, the strategies used to treat parasites in camelids must change. For many years, parasitologists have recommended that all animals in a herd be treated with an anthelmintic at the same time. This has proven unsustainable as there is still development of resistant parasites. The current approach is to selectively test and treat only animals with high parasite load, low body condition score (BCS), and pale mucous membranes. This will leave a population of parasites (refugia) that have not been exposed to specific drugs and will help prevent selection by the parasites for drug resistance. This also takes

into consideration that 20% of animals on a farm harbor 80% of the worms. Ideally, a fecal exam should be done on each animal before any anthelmintics are administered, which is impractical in a large herd, so monthly assessment of BCS and mucous membrane color can aid in choosing which animals to treat. As a minimum, 10% of the animals or at least 10 animals should be tested (various ages, etc) 2 to 3 times (or more) a year. If there are several barns, choose animals with low BCS from each with the total equaling 10 (or more). In addition, perform a fecal exam on every female immediately post-partum, and crias after weaning.

For this information to be meaningful, the correct procedure should be used. There are many different techniques and variations written about. An article by Cebra[1] et al comparing several techniques and floatation times concluded that the centrifugation-floatation technique (Modified Stoll's), using concentrated sugar (specific gravity = 1.27) and a 60-minute floatation time, was superior in detecting the parasites that have the potential for causing problems in camelids. A float time of 3 to 4 hours is also acceptable and preferred by the author (**not** 24 hours). It is important to be consistent for best results.

Mycoplasma haemolamae is a bacteria that acts like a blood "parasite" of camelids that can cause mild to severe anemia, but more often subclinical disease. Frequently the most common sign is just being thin. Clinical signs can vary from none to poor weight gain, generalized ill thrift, depression, and infertility. Clinical disease is seen most frequently if already immunosuppressed/stressed as it is considered to be opportunistic. If suspected, a complete blood count (CBC) with a spun packed cell volume (PCV) should be performed to verify degree of anemia and signs of regeneration. Whole blood should then be sent off for polymerase chain reaction (PCR) testing for *M. haemolamae*. Transmission is thought to occur from biting insects or blood contamination (needles, instruments, transfusion). It can also be transmitted *in utero*, and research is being done to determine the significance and occurrence of clinical disease from this form of transmission. Testing is best done by PCR on whole blood. The organism falls off the RBC after removal, so examination of a blood smear will frequently be negative unless the smear is made immediately and there are large numbers of circulating organisms in the blood. Treatment is oxytetracycline[c,d] (9 mg/lb [20 mg/kg]), SC, every third day for a total of 5 doses/injections (Bio-Mycin 200®[c] and Noromycin 300®[d] are preferred brands). In rare cases treatment may need to be continued if anemia still persists as treatment does not clear the infection, it just stops the replication of the "parasite".

Anemia is relatively common in camelids and can usually be attributed to 3 conditions discussed in this paper (gastrointestinal parasites, blood parasites, gastric ulcers) or anemia of chronic disease (these are just the most common causes). Lactating, chronically pregnant, and geriatric females tend to commonly have lower PCVs (19 to 22%) than reported as normal. These anemic animals should be evaluated as to the common causes and PCVs should be monitored routinely for change. Camelids are very resilient and do not readily show signs of moderate anemia. Routine monitoring of BCS and mucous membrane color is important to detect changes indicated and to allow measurement of PCV before serious debilitation occurs. In most situations the PCV needs to be below 8 to 10% (or 5%) before they are weak enough to be "down". Treatment is centered on stabilizing the circulation and replacing a portion of blood. During the lag time between catheter placement and acquiring blood from an appropriate donor, regular IV fluids plus steroids should be given for shock. Once stable, attempt to determine the source of the anemia and treat appropriately. See "Practical Fluid Therapy and Blood Transfusion for Camelids" in this conference *Proceedings* for complete information about fluid therapy and blood transfusion.

Meningeal worm infection is a disease in the midwest and eastern part of the United States caused by *Parelaphostrongylus tenuis*. The definitive host is the white tail deer and intermediate hosts are snails and slugs. Clinical signs can be seen 45 to 53 days after ingestion. Classic signs are rear limb weakness progressing to paralysis and involvement of front legs. Other signs can include an exaggerated gait, stiffness, overall weakness, circling, blindness, head tilt, seizures, and death. The signs can start suddenly or present with more gradual onset over months. No definitive test is available but cerebral spinal fluid (CSF) taps may show increased eosinophils and will rule out other possible causes (*Listeria*, meningitis). Frequently the diagnosis is based on response to treatment. Prevention is "deer-proof fencing", deep/wide gravel barrier on the outside of the fence to prevent migration of snails and slugs, and removal of dead fall, etc. to eliminate the favorable environment. Molluscicides can be used, however, be aware of possible contamination of water source. Guinea hens can also be used in pastures to eat the mollusks. Additional prevention is use of ivermectin[e] or doramectrin[f] in connection with the above listed management practices. Ivermectin or doramectrin should always be given SC as they are not effective for meningeal worm prevention if given orally or topically. Current dose recommendations are ivermectin 1.8 mL/100 lb (45 kg) SC, every 30 to 45 days or doramectrin 2.5 mL/100 lb (45 kg) SC, every 45 to 60 days. Treatment is most successful if done early in the course of the disease, and is directed at killing the larvae and controlling tissue damage. Treatment is fenbendazole[h] (23 mg/lb [50 mg/kg]) orally for 5 to 10 days plus flunixin®[g] (0.5 mg/lb [1.1 mg/kg]) SC, SID to BID for 4 to 5 days. If there is no response (unable to stand), then prednisolone (0.45 to 0.9 mg/lb [1 to 2 mg/kg]), IM or IV daily, can be given for 2 to 3 days. DMSO[i,j] (0.45 mg/lb [1 gram/kg] IV SID) can be given if no response after several days. One dose of ivermectin or doramectrin should be given to kill any migrating larvae still in the muscles. Physical therapy, slings and if possible, using a float tank can also be beneficial for recovery.

Polioencephalomalacia can occur in camelids, but very sporadically in the author's experience. Thiamine deficiency in the first compartment results in interference of the glucose pathway to the brain and subsequent softening of the grey matter. The cause of polio is generally undetermined, but usually associated with disturbance of C1 bacteria (feed change, stress, birth). On occasion polio will occur with overdosage of amprolium[k] because the drug will replace thiamine in the glucose pathway to the brain. Polio can also occur in some animals given sulfa drugs; the reason for this is unknown and is usually fatal as it does not respond to thiamine. The first clinical sign of polio is depression, but owners first notice blindness as the animal is found wandering aimlessly or walking into barn walls or feeders. In some cases, the signs progress to circling and seizuring. The recommended treatment is thiamine[l] (9 to 18 mg/lb [20 to 40 mg/kg]) SC or IV every 4 to 6 hours initially, continuing for several days after the clinical signs have improved and appetite has returned. If thiamine is given IV, it is best to add to IV fluids to avoid possible seizures. Start with the lower dose and increase only if the animal is not responding (still depressed, blind). Anti-inflammatories (flunixin prednisolone, DMSO) are also beneficial, as there is considerable inflammation. The author has encountered 2 different variations from the typical polio case. A few cases remained blind even though all other signs improved (appetite, depression) and treatment was initiated early. These same animals seem to regain some peripheral vision over months to years but cannot see things close up. They do very well in a group and can still raise a cria. The second variation is apparent early response to treatment, but quickly deteriorates into uncontrollable seizures and the animal has to be euthanized, with necropsy confirming the diagnosis of polio.

Listeriosis (*Listeria monocytogenes*) is a bacterial infection of the brain that occurs in camelids when on pasture or possibly from wet hay collected at the base of feeders. It is typically seen in younger, growing animals (< 5 yrs) from newly erupted teeth or **any** age from wounds in the mouth (stemmy hay). The bacteria travel up the trigeminal nerve in the gum and cause micro-abscesses in the brain. Clinical signs are typically confined to one side and include circling to one side, head tilt, drooping lip, nose deviated with a collapsed nostril, and unable to close an eye—all on the same side. This is not a common disease in camelids, but unless treated early and aggressively, it is usually fatal. Intravenous antibiotics are preferred, but an initial treatment attempt can be made with procaine penicillin G[m] (20,000 IU/lb [44,000 IU/kg]) SC twice daily, flunixin (0.5 mg/lb [1.1 mg/kg]) administered twice daily, and thiamine (9 mg/lb [20 mg/kg]) SC, 3 times a day. If over the next 2 days there is no improvement or the clinical signs become worse, the treatment should be switched to IV antibiotics. The author prefers ampicillin[n] (4.5 mg/lb [10 mg/kg]) IV 4 times per day or potassium penicillin[o] (20,000 IU/lb [44,000 IU/kg]) IV 4 times per day, but oxytetracycline at 9 mg/lb (20 mg/kg) IV SID can also be tried. Thiamine should be continued. Anti-inflammatories (banamine, prednisolone, DMSO) are also beneficial as there is considerable inflammation. In some cases, the clinical signs may improve to a point, but some residual signs may remain; however, most of the time they can still be a functional part of the herd.

Compartment 3 ulcers do occur in camelids; however, no studies have been done to determine the incidence and they may be less common than "diagnosed". As we are unable to visualize C3 with an endoscope, there is no definitive test for C3 ulcers. There are no typical changes seen on blood work or peritoneal fluid (unless an ulcer is ruptured). So diagnosis is usually based on clinical signs and presence of risk factors. Risk factors include stress from weaning, shows, transport or concurrent disease, use of non-steroidal anti-inflammatory drugs, feed changes, and reflux of bile from the duodenum into C3. Clinical signs can be colic, lack of appetite, depression, low fecal output, and on rare occasion grinding teeth and blood in feces. In the author's experience, although these signs may manifest, signs may be more subtle and consist of a cria or adult that apparently has a good appetite but is losing weight or has stagnant weight with mild to moderate depression. These animals should be evaluated for GI parasites and started on treatment for C3 ulcers. Use of ultrasound for further diagnostics is helpful to determine if there is an increase in peritoneal fluid and/or thickening of the wall of C3. Several products have been evaluated for treatment of C3 ulcers, with few able to work in camelids (ruminants). A study showed pantoprazole[p] is effective in inhibiting C3 acid production when administered to alpacas either IV 0.45 mg/lb (1 mg/kg, daily) or SC 0.9 mg/lb (2 mg/kg, daily), and is commercially available. After reconstitution (40 mg/vial) it is stable for 96 hours (per researcher, refrigeration preferred). Another drug used frequently as a gastro-protectant in other species is sucralfate.[q] Sucralfate given orally at 1 gram/50 lb (23 kg) BID – QID for 20 to 30 days is used frequently by owners with anecdotal reports of resolution of clinical signs of C3 ulcers. Although oral omeprazole[r] is ineffective as treatment of C3 ulcers, IV omeprazole (0.23 to 0.45 mg/lb [0. 5 to 1 mg/kg, IV]) every 6 hours has been shown to reduce C3 acid production for 6 hours; however, the IV formulation is available only from compounding pharmacies. With severe cases of suspected C3 ulcers, IV fluid therapy using partial parenteral nutrition in addition to pantoprazole (preferred product for treatment of ulcers) may be needed until appetite and demeanor improve. Force feeding a slurry may also be of benefit (contact Dr. Walker for recipe for nutritional supplement feeding instructions).

Hepatic lipidosis (fatty liver) should be considered in any camelid that is depressed, not eating, not acting like themselves, and may or may not have had sudden weight loss. It can be a sporadic problem and hard to diagnose, as blood work changes do not always correlate well with severity of liver disease. Specifically, look for increases (2-3X normal) in NEFA (non-esterified fatty acids), triglycerides, beta-hydroxybutyrate, cholesterol, and rarely hyperlipidemia.

Frequently hyperglycemia is also present, and this most commonly responds to resolution of the source of stress and hydration. If insulin is used to correct the hyperglycemia, it must be done with extreme caution and while on IV fluids. Frequent monitoring of blood glucose concentration is necessary as hypoglycemia will result in ~18 hours. Research has shown that camelids have poor response to insulin and are resistant to its effects. Treatment of hepatic lipidosis is based on reversing the negative energy balance (force feeding if necessary), flushing toxins from the system using partial parenteral nutrition, and restoring appetite. See "Practical Fluid Therapy and Blood Transfusion for Camelids" in this *Proceedings*.

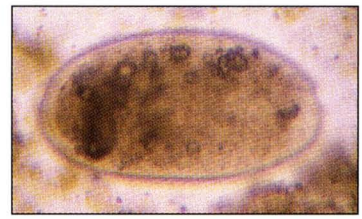
Viral causes of neurologic signs: Eastern Equine Encephalitis is seen mainly in the eastern United States, in late summer and fall and spread through mosquito vectors from avian hosts. Mammals are considered dead-end host due to low virus load in the blood. Camelids are at low risk for the disease, but it has been reported to be almost 100% fatal. Although there is some evidence that the equine vaccine can be used safely in camelids, it is only advised in areas of high risk and where there have been reported cases in horses.

West Nile Virus is seen across the United States in late summer and fall and is spread through the *Culex* mosquito. Clinical signs usually involve a twisting posture of the neck, uncoordinated gait, sensitivity to touch, inability to rise, and severe depression. Overall, camelids are at low risk for developing the disease; however, even with aggressive supportive care and treatment, there is a high fatality rate. The equine WNV vaccine can be used in camelids and does seem to be protective based on clinical response and research trials. It is recommended to give 3 injections 3 weeks apart in areas considered to be high risk.

Rabies has been reported in camelids in the United States. Clinical signs early in the course of the disease include lameness, uncoordinated gait, and paralysis in rear legs, similar to meningeal worm. This will progress to either an aggressive form or a paralytic form with death occurring within 1 to 4 days of onset of clinical signs. Encourage all owners to have a complete necropsy done on any animal that dies, especially with neurologic signs.

Balantidium coli is a large ciliated protozoan parasite found in swine and humans in third word countries. It has been reported in camels and has been seen in alpacas in the US. It is considered to be a normal inhabitant in the large intestine of the pig, and only rarely causes problems in that species. In alpacas, it seems to affect older animals most frequently and presents as moderate-volume, low-frequency diarrhea. It is a frequently overlooked cause of diarrhea. It can develop into a chronically intermittent syndrome, and over long duration can result in some loss in BCS. *B. coli* is transferred to host in the cystic stage. Transmission is fecal-oral by food and water contaminated with feces containing cysts. Cysts stay viable for weeks in the environment after passed in feces. Available information reports the trophozoite form survives for only hours in the environment, which is also the author's experience.

Most available information is not recent but does cover many species, including ruminants. It has been isolated from over 20 mammals including man, cattle, water buffaloes, and camels. In most situations, interaction with swine is connected with cases of balantidiasis in humans. However, in countries where there is no human association with swine, there are still cases of balantidiasis in humans. In those countries camels had the most interaction with humans, and trophozoites of *B. coli* were detected in 18% of feces of clinically normal camels. Their conclusion was it may be a normal inhabitant of the gastrointestinal tract of camels. However, there are a few reports of camels with *B. coli* trophozoites found in diarrhea and no other cause of diarrhea found, concluding in some instances it can be pathogenic. Clinical disease (diarrhea) is only considered to occur when the organism invades the lining of the intestines. This invasion is thought to occur when hyaluronidase is released and produces ulceration in the intestinal wall. This invasion may be aided by intestinal bacteria. Results of necropsies done on infected alpacas with chronic *B. coli* have damage that ranges from none, mild (most), to severe ulceration of the colon. Necropsy of positive animals may not always demonstrate severe damage unless the infection is chronic over many years.



Clinically in alpacas, *B. coli* cause medium-volume, low-frequency, intermittent diarrhea. The author has seen it in older imports and animals born in the US on several farms throughout the US. None of these farms have an association with swine or camels. In animals chronically infected, it seems to recur in the spring with change of feed by going out to pasture from a winter diet of dry hay. These animals are not becoming reinfected, it survives in the cyst form in the animal and the change of diet causes an imbalance in the GI tract and recurrence of diarrhea. The trophozoite form does not survive long out of the animal and will not be found in regular fecal floats. *B. coli* should be considered as a possible cause of diarrhea, especially when a sugar centrifugation fecal analysis does not show significant parasites including *E. mac*. As it does not survive long, so in order to diagnose a fresh sample of feces at the time of diarrhea must be examined immediately (within 1 hour or sooner) as a direct smear.

Treatments listed in the veterinary literature are no longer commercially available. In humans, treatment is oral tetracyclines or iodoquinol (anti-protozal). Camelids can be given oral and injectable tetracyclines, but drugs that kill protozoa will also kill off all the good protozoa in C1 and should be used with caution. The author has attempted different treatments on several farms. The current episode

will be shortened, but frequently the diarrhea will recur in animals chronically infected.

Treatments are focused on known safe anti-protozal drugs in ruminants: injectable tetracycline, oral tetracycline,[s] fenbendazole, and ponazuril.[t]

Diarrhea in adults can range anywhere from innocuous to serious. If it lasts for more than 1 day, especially with a depressed animal, further testing should be done. Testing should consist of some or possibly all of the following: fecal floatation, direct smear, viral EM, and bacterial culture. It is important to remember to do a direct smear on all cases of diarrhea looking for *Balantidium coli* trophozoites.

Endnotes

[a] Ivomec Plus®, Boehringer Ingelheim, Inc., 3239 Satellite Blvd, Duluth, GA
[b] Valbazen®, Zoetis, 10 Sylvan Way, Parsippany, NJ
[c] Biomycin 200, Boehringer Ingelheim, Inc. 3239 Satellite Blvd, Duluth, GA
[d] Noromycin 300LA, Norbrook, Inc., 9401 Indian Creek Parkway, Suite 680, Overland Park, KS
[e] Ivomec® 1%, Boehringer Ingelheim, Inc. 3239 Satellite Blvd, Duluth, GA
[f] Dectomax, Zoetis, 10 Sylvan Way, Parsippany, NJ
[g] Panacur®, Boehringer Ingelheim, Inc. 3239 Satellite Blvd, Duluth, GA
[h] Banamine, Merck Animal Health, 2000 Galloping Hill Road, Kenilworth, NJ
[i] DMSO – 106 Galeria Blvd, Slidell, LA
Caution: DMSO is an extralabel use, and must be used within the confines of a VCPR.
[j] Corid®, Pfizer Inc., 235 East 42nd Street, New York, NY
[k] Thiamine, AmerisourceBergen, 1300 Morris Drive, Chesterbrook, PA
[l] Procaine Penicllin G, Pfizer Inc., 235 East 42nd Street New York, NY
[m] Ampicillin, WG Critical Care, LLC, 120 Route 17 North, Paramus, NJ
[n] Potassium Penicillin G, WG Critical Care, 120 Route 17 North, Paramus, NJ
[o] Protonix, Pfizer Inc., 235 East 42nd Street New York, NY
[p] Carafate, Greenstone, 100 Rte 206 N, Peapack, NJ
[q] Gastroguard, Merck, 2000 Galloping Hill Road, Kenilworth, NJ
[r] Doxycycline, Epic Pharmacy, 5024 Campbell Blvd., Suite R Nottingham, MD
[s] Marquis, Boehringer Ingelheim, Inc., 3239 Satellite Blvd, Duluth, GA

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Reference

1. Cebra CK, Stang BV. Comparison of methods to detect gastrointestinal parasites in llamas and alpacas. *J Am Vet Med Assoc* 2008; 232:733-741.