

Megaesophagus in Camelids

BY DONNA ANDERSON



It was a rainy, dark Oregon evening and the transporter had finally arrived with Snowflake, a lovely suri girl—beautiful, dense fleece with lots of luster, a pedigree to die for, and excellently bred with a confirmed pregnancy.

A pre-purchase exam had confirmed that Snowflake was in excellent health. She had no problem conceiving and the veterinarian found no specific problems. Her health records as a cria indicated excellent weight gain, with 22 pounds at birth and 45 pounds at 30 days of age. The breeder did mention that Snowflake had, on occasion, regurgitated a small amount of green fluid. They had her checked by a camelid veterinarian and treated with antacid and probiotics for seven weeks. This was the only anomaly in our girl's otherwise excellent health history.

When Snowflake arrived, we placed her in our usual quarantine paddock and took a fecal sample to be tested by our veterinarian. She was found to have *Nematodirus*, so we treated her with Safeguard® for five days and at 14 days did another fecal exam. Now clear of parasites, Snowflake was introduced to our female herd, where she seemed to fit right in, showing good appetite and socializing well with the others.

One morning as I was making the “rounds” I noticed some thin green fluid in the paddock area where the girls sleep. It was not a large amount, but I immediately remembered what Snowflake's breeder had told me about her history. While I had not seen Snowflake regurgitate the fluid, upon inspecting her I noticed some green on her dense suri beard.

Our veterinarian was coming for a farm visit the following week, and I decided to ask him about Snowflake. I described her history and the fluid that I had observed. He did an exam and suggested that perhaps she was eating her food too fast. Since Snowflake seemed healthy and the problem did not seem to be severe, I decided to wait and see if it would go away. Snowflake was six months away from her due date and we had every reason to believe she would have a lovely cria from this excellent breeding.

As Snowflake's due date approached, I began to see the

spray of green fluid more and more frequently. I asked several other breeders if they had observed anything like this with their herds, but no one seemed to know what it was. I reviewed the archives on a couple of alpaca forums and could not find anything that seemed similar to Snowflake's condition. Optimistically, I was hoping it was something like morning sickness and would go away when she had her cria.

Finally, the day arrived—just like clockwork, her cria presented in a normal position with the two front legs and nose protruding. We observed the birthing process for another 20 minutes, being reluctant to step in too soon. But when it seemed as if Snowflake was having a little trouble getting the cria's head out, I scrubbed up, put on the OB gloves and used plenty of lubricant to place my fingers over the top of the cria's head and assist in getting it out. Birthing then proceeded normally.

Snowflake immediately recognized the lovely little fawn cria as her very own. She began making clucking noises, nosing and licking her little guy. It was a nice sunny day in October and the little cria was winking in the bright light so we nicknamed him “Winky.” Winky soon stood and shook vigorously, searching for his milk. We continued to observe him to make sure that he was nursing and getting that vital colostrum. All seemed good. Birth weight was 18 pounds.

However, on the second day of Winky's life, I arrived at the paddock to discover copious amounts of the green fluid, on the ground and on the cria. I cleaned Winky up and observed that he seemed to be nursing normally and strongly. But things were not as normal as I thought, and the situation deteriorated quickly.

By the afternoon of the second day, my husband observed bubbling of the milk around the cria's mouth and said, "Maybe the cria has the same thing as his mother." I began to worry that he was right. By the evening of the second day, the cria was coughing and did not seem so thrifty. Since it was after hours for our veterinarian, I decided to wait and see how Winky was doing in the morning.

On the third day, Winky was lying by himself, very still with his neck stretched out and eyes closed. I thought, "Oh my gosh, he's died." I reached down and picked him up by his cria coat and he just drooped. Then he wiggled and tried to stand. He seemed very weak. I immediately took his temperature and it was only 97 degrees Fahrenheit.

I wrapped Winky in a blanket and quickly drove to the veterinary office. Our veterinarian said that he had a respiratory infection and started him on the antibiotic Naxcel®. I decided to take Winky home and do my very best to save him, although I knew that the prognosis was not good. Using a heating pad, his temperature soon returned to normal but he was very weak and coughing. He took a bottle and consumed over four ounces of goat colostrum in the next few hours but was still getting weaker. Then he began to cough up blood. Winky died early in the afternoon on the third day.

This experience scared us. We knew it was time to take Snowflake to Oregon State University's College of Veterinary Medicine to see if they could figure out what she had, and whether there was a connection to Winky's problems. We checked her in at the Large Animal Clinic, where a veterinary intern began to take Snowflake's history as noted here:

Snowflake is a two-year-old female alpaca. She was purchased almost one year ago from a breeder that noted prior incidents of the regurgitation of green fluid. Previous examinations failed to find a cause for this condition. Snowflake has never been observed regurgitating, but pools were noted in the stalls she occupied and on other alpacas with increasing frequency. Snowflake had a cria four weeks prior to being admitted to OSU that died due to sepsis. Snowflake is fed pasture and orchard grass hay in addition to a vitamin and mineral supplement specifically formulated for alpacas.

Physical findings: Snowflake was bright, alert, and responsive on presentation. Her vitals were normal. She had a body score of 4 and weighed 133 pounds. Her oral membranes were pink and moist with a capillary refill time of less than two seconds. She had normal gastrointestinal sounds in all four quadrants. A holosystolic heart murmur was noted, along with an arrhythmia (skipped beats).

Diagnostic Tests: Plain radiographs of her cervical and thoracic regions revealed a possible dilation in the esophagus at the level of thoracic inlet. An esophogram with barium revealed pooling in the esophagus at the level of the thoracic inlet with possible dilation of the esophagus in the thorax.

Diagnosis: Megaesophagus.

Case Assessment: The cause for the esophageal dilation (Megaesophagus) is unknown in camelids. Clinical signs may be observed shortly after birth but are noticed more commonly at weaning. Signs commonly seen in esophageal dilation include hypersalivation and regurgitated feed that does not have the normal odor of first compartment fluid. Regurgitation occurs at any time, but may be seen more often when the head is lowered to eat or drink, or when the animal eructates (belches). Aspiration pneumonia can occur as a consequence. Megaesophagus is a progressive disease and prognosis is poor.

This was a very depressing diagnosis. I began to do as much research as possible and talked with other breeders about this condition. Should I keep Snowflake in the breeding pool? Is there any treatment? Does this occur in other species? Has any treatment been successful in other species? Is there a genetic connection?

What is Megaesophagus?

Merck Veterinary Manual has this to say about Megaesophagus: Moderate to severe dilation of the esophagus is relatively common in llamas and alpacas. Signs include chronic weight loss frequently associated with postprandial regurgitation or "frothing" of food. If the cervical esophagus is dilated, it is also sometimes possible to watch boluses of food move up and down the esophagus. There is no identified age or sex predilection, and no etiology has been established. A suspected case of Megaesophagus should be confirmed with barium contrast radiography and/or endoscopy. No treatments (surgical or changes in feeding practices) have been successful. The long-term prognosis is fair

to poor, with some animals maintaining condition for an extended period and others continuing to lose weight.

Since very little research has been done on this condition in camelids, I decided to look at its occurrence in other species. This is a condition that has been studied more extensively in canines.

“The esophagus is the tube connecting the throat to the stomach. When food is perceived in the esophagus, neurologic reflex causing muscle contraction and relaxation leads to rapid transport of the food into the stomach, like an elevator going down. Other reflexes prevent breathing during this swallowing process to protect the lungs from aspiration.

“When these reflexes are interrupted such as by disease in the esophageal tissue or nerve disease, the esophagus loses its ability to transport food. Instead the esophagus loses all tone and dilates. Also, the reflex protecting the lungs is disrupted and aspiration pneumonia commonly follows.

“Most cases are seen in young puppies (Great Danes, Irish Setters,

and German Shepherds are genetically predisposed). In these cases, the condition is believed congenital (present at birth) though it often does not show up until the pup begins to try solid food. Congenital Megaesophagus is believed to occur due to incomplete nerve development in the esophagus. The good news is that nerve development may improve as the pet matures. Prognosis is thus better for congenital Megaesophagus than it is for Megaesophagus acquired during adulthood.”¹

A 2010 article in the *Journal of the American Veterinary Medical Association*² suggests that camelids with esophageal problems that arise during the neonatal period may have a vascular ring anomaly, or VRA, and calls the prognosis “grave” in that event.

This was enough for me! Until we know more about this condition, it is my feeling that we should not keep breeding an alpaca with Megaesophagus, to reduce the potential for passing along something that might be genetically determined. Although I realized my impressions are unscientific, I believe

that Winky had a predisposition for megaesophagus and died from aspiration pneumonia. I have no absolute proof of this. A necropsy might have shown a VRA but we did not have one done.

Update: Snowflakes’s condition is progressively getting worse. I separate her from the herd at night so that she does not vomit on the other alpacas and I feed her a more digestible supplement. She is fed with an elevated feed bowl in an effort to get the nutrition where it needs to go. She is a beautiful girl and it is such a shame that she has this condition. Since her prognosis is so poor and she is getting worse, I am just putting off the inevitable. We will euthanize her when the time is right.

My thanks to Greg Fischer, DVM, of Newberg Veterinary Hospital in Newberg, Oregon for reviewing this article for accuracy.

Donna Anderson co-owns Double D Alpaca Ranch with her husband, David Carney. Donna and David both grew up in rural America and brought their love of the farming lifestyle to the alpaca industry when they began raising suri alpacas in Tigard, Oregon, six years ago. Over 30 alpacas now reside at Double D Alpaca Ranch. Donna can be reached at donna_anderson@doubledalpaca.com.

1. Wendy C. Brooks, DVM, DBVP, Mar Vista Animal Medical Center, on the Web site www.dogclinic.net, 2000.

2. McKenzie, E.C., et al, 2010, *Journal of the American Veterinary Medical Association*, Vol. 237, #3, “Esophageal dysfunction in four alpaca crias and a llama cria with vascular ring anomalies.”