Copper Deficiency and Alpaca Disease, a Case Study

BY SHEILA SCOGGINS

How can we uncover any emerging or re-emerging disease in New World camelids unless we look for the cause of death when we lose an alpaca? This dilemma ultimately faces all owners at some point when raising alpacas.

Finding the reasons for a death is good for our camelid medicine, and it is good for each owner, even if just for curiosity reasons, not to mention to ensure we are not dealing with a contagious disease.

For me, the most important reason to determine cause of death is to add to the body of medical knowledge on alpacas. The cost is well worth it. There could be diseases emerging in the alpaca, diseases seen in other species, but not yet seen in the camelid. This is a case report about a well-known disease, and a not-so-well-known disease. As you will see, even when the cause of death is determined, you can be left with more questions than answers.

During the summer months we lost two crias unexpectedly. The two crias died less than two weeks apart. The first death came with a 3-month-old cria, and the second was 4 months of age. Both deaths came in what we thought were healthy crias. Cria 1 was born healthy and had normal growth and development up until her death. Unbeknownst to us, she had a serious disease and an even a more serious mineral deficiency. The final pathology report (Figure 1) showed severe interstitial pneumonia, bronchopneumonia, and severe mineral depletion of copper, zinc and selenium.

The UC Davis California Animal Health and Food Safety pathologist's opinion was the interstitial pneumonia could represent a pneumonia first identified in the U.S. in alpacas in 2007. Breeders called it "the snots." In the 2007 outbreak, the corona virus termed the ACoV was postulated to be the causative organism. (2)

There were a number of cases of "the snots" in 2007, and then the rates of the infection dropped away significantly. Because of symptoms similar to "the snots," Cria 1 was tested for ACoV. Unfortunately, a definitive answer was not to be. Cria 1 was negative for the corona virus.

To make the diagnosis even more confusing, the cria had a large neck and shoulder hemorrhage. The hemorrhage led to thinking maybe Cria 1 had been

FINAL NECROPSY FINDINGS CRIA 1

1. **Bronchopneumonia, neutrophilic, acute; Bordetella sp.**
2. **Interstitial pneumonia, severe, fibrinous (hyaline membranes), multifocal necrotizing and neutrophilic**
3. **Hepatic centrilobular degeneration, mild (vacuolation)**
4. **Adrenal cortical cellular vacuolation (lipidosis).**
5. **Tonsilitis, focal necrotizing with intralesional bacteria.**
6. **Copper deficiency, severe.**
7. **Selenium and zinc deficiency.**
8. **Right side of neck and shoulder: muscle hemorrhage (friable)**

Figure 1: University of California at Davis necropsy report on Cria 1

Background photo by Jennifer Clark

Cria Aphrodite was apparently healthy prior to her death.

Photo courtesy of the author

bitten by a venomous snake. All of the other findings would and could be a result of venom, so the final and most likely conclusion was a rattlesnake bite. For a short time we thought we had just had bad luck.

Shocked by all this horrific information about Cria 1, an alpaca we had thought was healthy, we worried about the possibility of a deadly infectious disease and whether we had rattlesnakes on the property. How were we going to know if we had "the snots," given there seemed to be no warning signs? And how could we have such a severe mineral problem? The necropsy report gave us at least three ways Cria 1 could have died. We were going to have to deal with all of them to protect the rest of the herd.

Death of second cria complicates diagnosis

Then we lost another cria. When Cria 2 was found dead, our worry went to terror. Cria 2 was small for gestational age, but just like Cria 1, appeared healthy prior to death.

When we delivered Cria 2 to the CAHFS lab at UC Davis, they and we knew the problem had now risen to a much higher level. The first request we made was to determine if the cria had a hemorrhage. If both animals had been poisoned we would need to find out how. The pathologist, bless his heart, called us within four hours: No hemorrhage.

The bad news: Cria 2 also had severe broncho-pneumonia. We were frantic but baffled. How could

Table 1: Levels of heavy metals detected in Cria 1.

CAHFS Final Version – HEAVY METAL SCREEN – LIVER TISSUE					
MINERAL	**RESULT**	**UNITS**	**REP. LIMIT**	**UNITS**	**REF. RANGE**
		CRIA NO. 1			
Lead	Not Detected	PPM	1	PPM	<2.0
Manganese	**0.11**	**PPM**	**0.04**	**PPM**	**2.0-4.0**
Iron	210	PPM	0.2	PPM	70-200
Mercury	Not Detected	PPM	1	PPM	<1.0
Arsenic	Not Detected	PPM	1	PPM	<2.0
Molybdenum	Not Detected	PPM	0.4	PPM	<1.0
Zinc	**12**	**PPM**	**0.1**	**PPM**	**20-90**
Copper	**0.71**	**PPM**	**0.1**	**PPM**	**25-100**
Cadmium	Not Detected	ppm	0.3	PPM	<1.0
Selenium	**0.16**	**PPM**	**0.020**	**PPM**	**0.25-1.0**

we know who was ill, if there were no obvious symptoms, and how could we prevent more deaths? Were we going to lose all our spring babies? What about our pregnant girls, all were in the same pasture?

One problem we knew we had to correct was this mineral problem. We definitely needed advice.

Copper deficiency is a known cause for immunocompromised alpacas. (1, 8, 9) Immunosuppression makes the alpaca susceptible to infectious disease. Though Cria 1 had more than just a copper deficiency, the pathologist put more emphasis on the lack of copper influencing the cria death. The pathologist thought Cria 1 definitely was more susceptible to pneumonia due to the lack of copper.

Cria 1's mineral deficiencies were profound. Much is written about selenium and zinc deficiency and little about copper, yet, copper deficiency is extremely dangerous. The role of copper in alpaca physiology is diverse. Copper has influences in iron regulation, oxygen-carrying cell functioning, bone and connective tissue formation, nerve and cardiac cell functions, and immune function. (1, 8)

Our animals definitely needed mineral testing. We had to determine the extent of the problem with our other babies. We have always been careful to give all the animals their daily mineral supplement, so, Cria 1's necropsy results really confused us.

Our veterinarian recommended we test all crias and their dams, which we did. Unfortunately, the results made for even more confusion. One dam and cria had copper deficiency, and another had no deficiencies. Fortunately, none had zinc or selenium deficiency. To make for even more confusion, the necropsy on Cria 2 results (Table 2) showed no mineral deficiency. Good to hear, but the results seem to make determining the extent of the mineral problem even more of a conundrum.

Of all the minerals, Copper (Cu) has the greatest influence on fiber if depleted. The lack of copper creates fiber with color losses and loss of fineness

Table 2. Results of heavy metal screen on Cria 2.

HEAVY METAL SCREEN – LIVER TISSUE					
MINERAL	**RESULT**	**UNITS**	**REP. LIMIT**	**UNITS**	**REF. RANGE**
		CRIA NO. 2			
Lead	Not Detected	PPM	1	PPM	<2.0
Manganese	2.4	PPM	0.04	PPM	2.0-4.0
Iron	170	PPM	0.2	PPM	70-200
Mercury	Not Detected	PPM	1	PPM	<1.0
Arsenic	Not Detected	PPM	1	PPM	<2.0
Molybdenum	0.75	PPM	0.4	PPM	<1.0
Zinc	46	PPM	0.1	PPM	20-90
Copper	55	PPM	0.1	PPM	25-100
Cadmium	Not Detected	PPM	0.3	PPM	<1.0
SELENIUM	0.81	PPM	0.020	PPM	0.25-1.0

Table 3. Results of trace mineral screen on blood from Dam 1, Cria 3 and Dam 2

CALCIUM	**COPPER**	**IRON**	**MAGNESIUM**	**ZINC**	**SELENIUM**	
Ref. Range:	80-100	0.4-1.0	0.7-1.5	18-35	0.3-1.5	0.08-0.5
Unit:	PPM	PPM	PPM	PPM	PPM	PPM
Result:	Result:	Result:	Result:	Result:	Result:	Result:
Dam 1	73	0.24	0.71	19 0	.74 0.	19
Cria 3	88	0.19	1.4	17	0.34	N/A
Dam 2	85	0.36	1 24	46	0.36	N/A

A necropsy done on Cria 2, Orion, mostly served to confuse the issue of what the problem was.

Photo courtesy of the author. Background photo by Jennifer Clark

and crimp. In fact, Dr. Van Saun of Penn State talks about copper deficiency fiber being described as harsh "brillo" like fiber.(8) Of the diagnostic findings on five alpacas, three had copper deficiency – not what you want to see in crias expected to be show animals.

Is Bordetella bacteria part of the problem?

Cria 1 did have a positive lung culture result. The culture result showed Cria 1 had Bordetella bronchiseptica. While you may not be familiar with the Bordetella bacteria species in humans, you might be familiar with the disease, which causes Pertussis, more commonly known as Whooping Cough. Bordetella bronchiseptica is also the causative bacteria in kennel cough in dogs. B. bronchiseptica is a common disease-producing bacteria. It is treatable with antibiotics; often it needs no treatment and the animal fully recovers. That is, of course, if the animal is not immuno-compromised, when a benign illness can become a killer.

Cria 2's lung culture results were negative for any bacteria. The necropsy report for Cria 2 did indicate that the lung appearance was similar to that of Cria 1, even though no bacteria was found. It is possible to get a negative culture result even when a serious infection is present. Hence, cultures can definitely rule in a pneumonia, but a negative culture cannot rule it out.

In all likelihood, Cria 2 also had Bordetella bronchiseptica. The problem with Cria 1 and probably Cria 2, is neither had the distinct symptoms, such as a cough, expected with a Bordetella bronchiseptica infection.

Besides B. bronchiseptica bronchopneumonia, Cria 1, with a high degree of suspicion, was believed to have "the "snots." Though the name "snots" makes it sound minor, the disease is not. The appearance and condition of the lungs on necropsy were consistent with Adult Respiratory Distress Syndrome (ARDS). ARDS is quite serious and carries a high death rate. (6) ARDS is always a complication of another disease. (6) Critical injury or illness can result in ARDS in just about any animal, including humans. Hence, the pathologist felt the B. bronchiseptica was not severe enough to cause the ARDS. She believed the most probable cause of ARDS was by the "snots" pneumonia.

The pathogen believed to cause "snots" is the Corona Virus (ACoV).(2, 11) To give you an idea how bad this virus can be, the human SARS epidemic in the early part of 2000, was caused by a Corona Virus. (2, 10) In literature search, and in talking to this pathologist, the means of transmission of the "snots" virus is still not completely understood. It makes you wonder if this ACoV virus is now re-emerging in 2015. Or, is it simply being missed or misdiagnosed? Like the Bordetella bacteria, the ACoV virus does not always show up on diagnostic tests. (11) Cria 1's blood test for the ACoV PCR was negative, but the lungs looked just like lungs with the "snots." There are so many questions about this virus, yet no one really has the answers.

Neither cria had evidence of sepsis. Dave Warshauer 's research indicates that B. bronchiseptica rarely causes sepsis, is found primarily in immunosuppressed animals and rarely is isolated from pulmonary

tissue.(1, 2, 9) Again, it is probably safe to assume cria 2 also had B. bronchiseptica, regardless of the negative culture.

In the research for this article, I found little to no specific information on B. bronchiseptica in camelids. There are significant numbers of research articles for B. bronchiseptica in other animals. I don't know if this is reflective of the rarity of this disease in camelids, or that it goes undiagnosed and there is under reporting of the disease. (1) Definitely, more medical research is needed in the prevalence and incidences of this infection in new world camelids.

What Happened Next

We did have four animals become mildly ill after the identification of pneumonia in Cria 1's necropsy. Most B. bronchiseptica infections in other species are caused by outbreaks from shows, or, where animals are in close proximity, such as dogs kenneled together. None of our alpacas had contact with outside alpacas since showing in February. So then, how did crias 1 and 2 contract B. bronchiseptica? We have no answer to this questions.

At the direction of our veterinarian, we instituted strict surveillance with all alpacas housed with crias 1 and 2. There were 17 alpacas, of which four were crias. Of those, four became mildly ill and were moved into isolation. Two of those ill animals were tested, and, were confirmed to have copper deficiency. Of the four ill animals, the two with the copper deficiency were the more clearly symptomatic.

The symptoms of animals we considered ill were signs easy to miss. We only detected the illness because we were physically examining all of these alpacas twice a day. The surveillance consisted of temperature, weights, lung sounds, nasal discharge, mucous membrane, heart rates and respiratory rates.

Of the four ill alpacas, the four consistent signs we encountered were:

- very slight nasal discharge (some of the time we were not even sure it was mucous versus moisture from the pastures).
- low-grade fever (102.8 to 103.5).
- harsh lung sounds (no adventitious lung sounds).
- increased respiratory rate.

As part of the surveillance we would also just "eyeball" every animal to see if they were eating and looking bright. Interestingly, none of the ill alpacas had weight loss. Plus, the ambient temperature was in the mid- to high-90 degree range. As you can see, the symptoms were quite mild and easily overlooked. No animals exhibited any cough, lethargy or went off their feed.

The literature indicates a high degree of resistance of Bordetella species to some antibiotic. (3, 4, 5) Cephalosporin antibiotics are indicated as there is less resistance to them. (3, 4, 5) Ceftiofur dosing was initiated at 1 milliliter per 22 lbs. once daily. We needed to "up" the dose on one cria and two adults who spiked temperatures again after two days of therapy. Each of the four alpacas gradually improved.

More Questions than Answers

As worthwhile as the necropsies were to help treat our animals, they provided more questions than answers. Was Cria 1 so undermined by the copper deficiency that she developed the bronchopneumonia and "the snots"? But if that is the case, and "the snots" is contagious, as is Bordetella, why did Cria 2 not succumb to the same "snots" and ARDS. Why did this particular cria develop such a severe copper deficiency? Maybe the copper deficiency is the key to Cria 1's more debilitated state. Regardless, both crias had a pulmonary disease sufficient to cause death.

I am certainly hoping we are not seeing a resurgence of ACoV-induced interstitial pneumonia. Or, are we beginning to see a new infectious disease in alpacas caused by Bordetella bronchiseptica? I do know I will be much more conscientious to insure every alpaca eats their daily rations of their alpaca pellet supplements. We now have a backup source of minerals in every pasture.

So here we are, ending up with more questions than answers. My advice to other owners would be: don't take mineral health in your alpacas for granted, even when using supplements and especially for the levels of copper. Copper deficiencies could be more prevalent than most think. I will continue to promote the use of necropsy to learn about our alpacas, not just for my learning, but also for the entire alpaca community. Let those alpacas we lose teach us.

I want to thank our veterinarian for all his help and guidance with this health issue, and also thank the pathologists of the CAHSF U.C. Davis lab for helping our farm. Their support and guidance is much appreciated. Their keen expertise was abundantly evident. ■

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