The Perfect STORM

stock.xchg.com

Case Study of a Septic Cria

BY SHEILA COONEY SCROGGINS

This case study is about Moon River, a beautiful alpaca cria that was born healthy. She appeared by all measures to be normal, but lived for only four short days. Moonie, as we nicknamed her, succumbed to an infectious process, the result of insufficient amounts of colostrom in the first hours of her life. All this was unbeknownst to her owners, until it was too late. My hope in writing this case study is that you develop an understanding of the consequences for a cria who fails to get enough colostrom in the first 12 hours of life.

"Sepsis is a severe illness in which the bloodstream is overwhelmed by bacteria".[1] The more accurate diagnostic consequence of sepsis is what is termed Systemic Inflammatory Response Syndrome (SIRS). Regardless of the animal SIRS afflicts, the bacteria must have an entry point into the bloodstream and the animal must be vulnerable. In the newborn cria, the entry into the bloodstream is usually the umbilical cord. Invading bacteria also need a susceptible host.[2] In the newborn cria, that immune vulnerability is produced when the cria gets inadequate amounts of colostrom, or poor quality colostrom.

Camelids are born with a condition called, in medical terms, agammaglobulinemia. This simply means they are born with no passive immunity acquired during gestation. During the pregnancy, the gestating cria attains no temporary source of immunity to common environmental bacteria through the placenta. The entire source of the camelid immunoglobulin (IgG), or temporary immunity, is acquired from the colostrom the cria ingests in the hours after birth.[3] The newborn camelid cria's immunity to infectious agents is completely dependent on receiving adequate and early doses of colostrom.

An easy way to think of passive transfer immunity is that all cria come in contact with common bacteria from the minute they hit the ground. The dam protects her cria from these common bacteria by supplying the cria with her immune cells' knowledge (IgG) of the local microorganisms. Since contamination is inevitable in the first hours of life, the dam passes antibodies that recognize bacteria until the cria can produce enough of her own antibodies. The ultimate consequence of a failure of passive IgG transfer through colostrom is the cria's immune system has a poor recognition of the most common of environmental bacteria.

Photos courtesy of the author.

Left, the calm before the storm: Moon River nurses from her dam, Sundance.

Right, Moonie was a large, healthy cria at birth, before sepsis set in.

Authors Garmendia, Palmer, DeMartini, and McGuire provide us with some statistics on just how much this lack of passive transfer of immunoglobulin can influence the mortality of newborn cria. Garmendia, et al, studied 82 crias. Of those 82, 10 died within two months of age. Of those 10 that died, seven had lack of passive transfer of immunologlobulin.

Of those seven with lack of IgG, five had evidence of infections. This study also found that the lower the serum IgG level in the cria, the poorer the prognosis and the higher the incidence of mortality.[4] More importantly, without supplementation, either by an alternative colostrom or transfusion, the prognosis for most cases of failure of passive transfer is very poor.[5]

Colostrum ingestion is a simple natural act. However its absence creates multiple problems for the newborn cria. Passive transfer of IgG is extremely time-sensitive. After 12 to 24 hours the cria gut is believed to "close." A simplistic explanation of this "closing" is that the gut loses the receptors which allow for transport of the ingested antibodies across gut wall and into the bloodstream. This absorption window is believed to drop off precipitously by six hours after birth.[4] Once the gut "closes," all the ingested colostrom in the world won't make it into the cria's bloodstream.

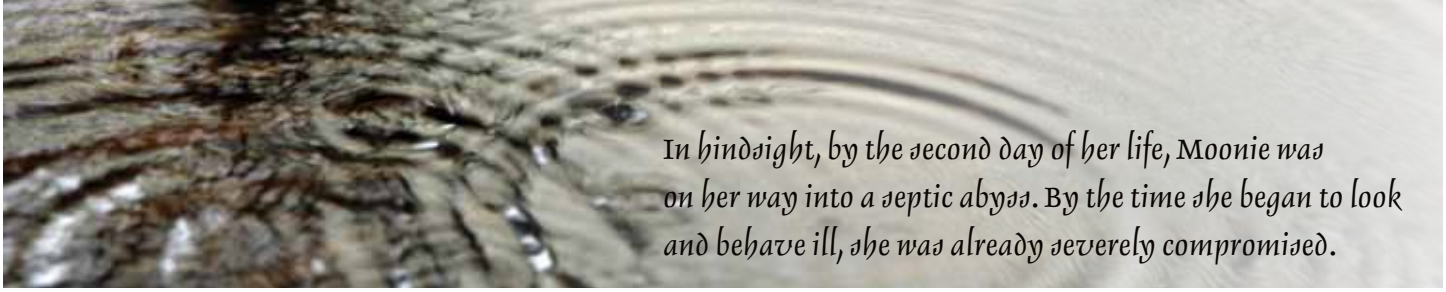
In our case, and in hindsight, by the second day of life, Moonie was on her way into a septic abyss. By the time she began to look and behave ill, she was already severely compromised.

References vary on the time in which the gut closes, but all agree if suckling is delayed, you need to intervene quickly.[5] An owner's most critical assessment of the newborn cria is how much, how often and how vigorously the cria nurses during the first six hours. Dr. Pamela Walker, in her 2010 Camelid Symposium presentation, spoke to the need to assess the cria's suckling every hour in the first eight hours. Dr. Walker further recommends an intervention at three hours if this sucking does not proceed often and with vigor.[6] Some references recommend as early as two hours if suckling is inadequate.

If suckling is deemed to be inadequate, owners must get colostrum into the cria from other sources. Milking the dam would be the optimum choice, if possible. Dr. Scott Haskell, Program Director of the Yuba College Veterinary Technician Program, recommends using the first milking of bovine colostrom as a supplemental colostrom. The bovine colostrom, of course, must be free of BVDV, Johnes, and other infectious agents.[7] Some alpaca owners supplement with bovine colostrom regardless of the cria's nursing history.

Alpaca colostrom is also a high energy source for the newborn cria. The native environments of camelids dictate their colostrom must contain high sources of carbohydrates necessary for the conditions

1. Duey, B., & Duey, S., 2005, "Passive Immunity Transfer and Plasma Transfusions for Alpaca Cria, Husbandry Hints", *Alpacas Magazine,* Herd Sire 2005.
2. Forbes, B., Sahm, D., Weissfeld, A., 1998, *Bailey & Scotts Diagnostic Microbiology,* Mosby, Inc., 11th ed.
3. Tibary, A. and Anouasssi, A., 1997, *Theriogenology in Camelidae; Anatomy, Physiology, Pathology and Artificial Breeding,* United Arab Emirates, Ministry of Culture and Information, Abu Dhabi Printing and Publishing Co., 1st ed.
4. Dunlop, R.H. and Malbert, C. H., Eds., 2004, *Veterinary Pathophysiology,* Blackwell Publishing.
5. Barrington, G., 2008, *Blackwell's Five-Minute Veterinary Consult Ruminant,* John Wiley & Sons, Ltd. Publication, 1st ed., and also Tibary, A., and Anouasssi, A., 1997, *Theriogenology in Camelidae; Anatomy, Physiology, Pathology and Artificial Breeding,* United Arab Emirates, Ministry of Culture and Information, Abu Dhabi Printing and Publishing Co., 1st ed.
6. Walker, P., DVM., 2010, "Neonatal Care of Camelid Crias", Guest Lecturer, Camelid Symposium.
7. Haskell, S., DVM., 2010, Personal Interview, Yuba College, Marysville, CA.



In hindsight, by the second day of her life, Moonie was on her way into a septic abyss. By the time she began to look and behave ill, she was already severely compromised.

under which they are born.[4] Alpaca colostrom is also high in fat and protein concentrations, which provide the cria with complex, long-acting energy sources. If the cria gets insufficient colostrom, its blood sugar levels can drop too low to provide the energy necessary for the cria to continue to nurse adequately. As we would later find out through laboratory tests, Moonie had significant drops in blood levels of glucose.

Moonie had a classic case of failure of passive transfer. But if that was not insult enough, she was born during the hottest week of the summer of 2010. Moonie became heat stressed and by the time we reached U.C. Davis with her, she was severely dehydrated.

Moonie was a large cria, weighing over 20 pounds at birth. Her gestation was 372 days. All of these factors—her size, the long gestation, and the heat—contributed to a difficult birth. But once she was on the ground, her size initially lulled us into a sense of security and a bit of denial that went on too long.

We did not witness Moonie's birth, but it was clear to us that it was difficult. Afterwards, her dam was depressed, she had a stiff gait, and had difficulty urinating and defecating. The dam was reluctant to nurse Moonie unless medicated with phenylbutazone (sometimes known as "bute") for pain. All of these factors contributed to a cria that was nursing poorly and a dam with poor colostrom production. Events came together to overwhelm this newborn cria and cause her demise within only four days of birth.

When reviewed, the course of Moonie's deterioration is clear. The first few hours of life seemed uneventful; she followed her mom appropriately and made many attempts to nurse. However, by twelve hours after birth, she had begun to show subtle signs of decline. Her level of activity gradually decreased. When her dam walked to the dung pile, Moonie stopped following.

By day two, I knew I had a problem, so I began to supplement Moonie with colostrom and milk. Moonie voraciously suckled the bottle at the start, and we continued to supplement her over the course of day two. Even with the supplementation, though, her lethargy persisted. Most disturbing of all, Moonie began to exhibit behaviors consistent with what is called a "Dummy Cria." For example, she would walk over to a tree, take a cush position, and place her face up against the trunk. Her dummy cria symptoms could have been from lack of oxygen during birth, but they just as easily it could have been from her early dehydration or lack of serum glucose.

Moonie had lost a pound between day one and day two, but she appeared to be nursing well when she did nurse. One pound was concerning, but not completely out of the ordinary. We moved mom and cria into the barn under a large fan, and continued with frequent supplementation.

On day three, we weighed Moonie early in the morning and she was down an additional two pounds. This represented nearly a 15 percent loss in body mass and fluid, an indisputable sign of severe dehydration, and one that rapidly occurred over the course of just the prior 24 hours. While Moonie's temperature was not elevated,, her respiratory rate and heart rate were at the upper ends of normal. I knew she was in grave trouble.

We loaded mom and cria and headed for UC Davis Veterinary Hospital. By this point, we had a cria that was very weak, ataxic (uncoordinated) and lethargic.

Moonie's first blood work was done after admission to Davis's Neonatal Intensive Care Unit. Her blood glucose was 33 milligrams per deciliter (mg/dl). Normal glucose in the newborn cria should be 80 to 140 mg/dl. It was not until six hours later and significant amounts of intravenous dextrose that Moonie's blood sugar rose to a near-normal value. It is likely that after birth she had been suffering from, and exhibiting clear signs of, hypoglycemia or low blood sugar. The blood sugar value of 33 mg/dl is a dangerously low value and sufficiently low to reduce the metabolic functions essential for life.

As other diagnositic studies were completed, it began to emerge just how sick Moonie was. Most telling was her blood immunity picture. She had a total White Blood Count (WBC) of 2,000. The normal WBC in a healthy cria should be greater than 8,000.[5] Moonie was neutropenic (having an abnormally low level of neutrophils, the most abundant type of white blood cells) with a neutrophil count of only 36 percent. Her immune cells were being used up quickly by an evolving, overwhelming sepsis. By this point, Moonie's temperature was elevated and her pulse was excessively high. A septic cascade was taking hold, a sequela of the events of her first hours of life.

Dr. Norm Evans, in his work *"The Alpaca Field Manual,"* indicates an elevated monocyte (another type

of white blood cell) count can be a significant indicator of alpaca stress. Evans states that an 8 percent to 10 percent increase in monocyte cells in the alpaca "equals major problems in the animal's health."[8] Moonie's monocyte count had increased by 9 percent. The evidence of Systemic Inflammatory Response Syndrome also began to emerge. Dr. Evans further describes clear evidence of serious infection is the finding of a serum fibrinogen level above 350 mg/dl. Moonie's fibrinogen was 600 mg/dl. Coupled with her WBC, the evidence was increasingly clear that we were dealing with SIRS, as a result of sepsis. The deadly consequence of SIRS is organ damage, in particular, damage to the kidneys and lungs, both of which later became evident in Moonie.

Moonie's state of dehydration was easily identified in the results of her blood work and by her weight loss. Her blood volume was so depleted due to dehydration that her serum salts were well above normal concentrations. Her kidneys had sustained damage. She had serum elevations of blood urea, nitrogen and creatinine, definitive evidence of a serious insult to the kidneys. At this point, it was unknown whether or not the damage to the kidneys was reversible.

Even with all of Moonie's abnormal manifestations and poor diagnostic findings, we were given a 50 percent chance of recovery. With this prognosis, we asked the veterinarians to continue her treatment for the next 12 hours and then we would re-evaluate her situation. Everything was riding on whether her organ damage, infection and dehydration were reversible.

Moonie was treated over the day and night with two plasma transfusions, a number of IV fluid boluses, antibiotics, oxygen, anti-inflammatory drugs, and urinary catheterization. At one point during the night, she was bright and nursing with some vigor, giving everyone hope.

Then as quickly as she brightened, she began another vicious decline, and this time it was rapid. When we arrived in the morning, she could no longer hold her head up without help. She was having marked difficulty breathing. With this new onset of symptoms, we knew she was now suffering from another compromised organ system. Her lungs were filling with fluid, or she had pneumonia.

We were now dealing with two severely compromised organ systems. While Moonie's hypoglycemia and dehydration had improved, she was possibly in respiratory failure. The only choices were to give up entirely or get even more aggressive in her treatments. With these new manifestations, her prognosis was downgraded. We now had our answer. My medical knowledge and experience began to overwhelm my decision. I had witnessed a number of patients succumb to SIRS with one organ system failure after another. I knew I had a chance of saving her, but for what? I knew that to save her would have a medical cost I could not sustain. The decision was made; Moonie would be euthanized.

Lack of passive transfer was the start—the trigger—for Moonie's problems. Her necropsy report justified our decision to euthanize. The report showed significant amounts of immature immune cells in multiple organ systems, indicative of an infectious process and the response of an underdeveloped and inadequate immune system.[9] Our decision to end it was wise.

Failure is always going to be a part of raising alpacas, but failure that ends in the death of an initially healthy cria is by far the most difficult. Writing this article has been difficult, but I hope by reading it, others will gain understanding enough to recognize the symptoms of failure of passive transfer. By sharing our experience with Moonie, we hope to help you save one of your own crias someday.

The author would like to offer special thanks to Dominic Dawson DVM, DACVIM for the care of Moonie, but also for her contributions to this article, and a special acknowledgment to Scott Haskell DVM, MPVM, PHD, a colleague and resource for everything veterinary, and a contributor to this article.

About the Author: Sheila Scroggins is the owner of Colusa Riverside Alpacas of Northern California. Sheila is a Nurse Practitioner who has been practicing nursing for over 30 years. She is Professor of Nursing for Yuba College, and presently is Director of Nursing. She invites you to visit her web library at www.colusariversidealpacas.com and read her other publications.

8. Evans, N., D.V.M., 2009, *The Alpaca Field Manual.* 3rd Ed.

9. Koehne, A. L., DVM. (2010). Pathology Necropsy Final Report. Veterinary Medical Teaching Hospital, University of California, Davis.