W 781

Bovine Anaplasmosis

Amber Moore, DVM, MS
Lew Strickland, DVM, Extension Veterinarian
Department of Animal Science

Introduction

Bovine anaplasmosis, caused by *Anaplasma marginale,* is a rickettsial disease characterized by progressive anemia. It is the most prevalent tick-borne cattle disease in the world and accounts for $300 million lost to the U.S. beef industry annually (Whitlock, 2014). This bacteria is also able to infect sheep, goats and some wild ruminants. Although the bacteria infect these animals, they do not typically show clinical signs and act as reservoirs for the disease. Many areas in the United States are endemic for this disease and see significant economic losses in both the beef and dairy industry. Diagnostic labs across the state have reported an increase in the number of anaplasmosis cases they see from year to year. Possible reasons for this include increased cattle movement due to drought or natural disaster and increased movement of ticks that harbor the bacteria.

What causes anaplasmosis?

Various species of anaplasma affect most species of animals including humans, dogs, horses and cattle. Bovine anaplasmosis is most commonly caused by *Anaplasma marginale*. This disease is typically found in tropical and subtropical regions, including the southern United States with a prevalence ranging from 2 percent to 24 percent (Whitlock, 2014). The incubation period ranges from one to eight weeks, with natural infections occurring within three to five weeks. This is dependent on the amount of exposure the animal receives. The bacteria is introduced into the bloodstream where it is replicated in the red blood cells (RBCs). In an effort to clear the infection, the immune system removes and destroys these infected RBCs causing an extravascular hemolysis and characteristic progressive anemia. Between 10 percent and 30 percent of the RBCs can be infected at the peak of the disease (Lew-Tabor, 2018).

Animals that survive the initial stages of the disease become chronically infected. The animal remains a carrier of the bacteria for life and is immune to clinical disease. This becomes problematic when carriers are mixed with naïve animals. It is not known where the infection persists within the body; however, the blood remains infected even if the bacteria cannot be found on a blood smear. If carrier animals ever undergo immune suppression due to steroid use or infection with other pathogens, they can relapse and show the listed clinical signs.

The severity of the disease is related to the age and breed of the animal. Calves less than 1 year old tend to be more resistant to clinical disease than older cattle. These calves will have no to minimal clinical signs and become silent carriers. Cattle up to 2 years of age present with acute infection that is rarely fatal and become carriers. Adult cattle are the most severely affected and present with severe, acute and potentially fatal disease. In adult cattle that are naïve to the disease, there is a 3.6 percent less successful calving, 30 percent increase in culling rate, and cattle experience a 30 percent mortality rate (K. Pompo, 2016). *Bos taurus* cattle (Angus, Holstein, Hereford, Charolais) are more likely to develop severe disease than *Bos indicus* cattle (Brahman, Gyr, Nelore). The reasons for the age and breed susceptibility differences are unknown.

How is anaplasmosis spread?

A. marginale is most commonly transmitted from carrier to susceptible cattle via arthropod vectors. Several tick species have been found to carry the bacteria. These ticks include *Dermacentor* (American dog ticks), *Rhipicephalus* (brown dog tick) and *Ixodes* (blacklegged ticks) species. *Dermacentor* is thought to be the main vector within the United States. The bacteria is able to replicate within the tick and can remain in the ticks through life stages.

Horse flies and stable flies act as mechanical vectors and require close proximity of infected animals. Literature also states that mosquitos can act as vectors; however, their role in spreading the disease is minimal unless there is a severe infestation.

The second mode of transmission is iatrogenically through contaminated needles, dehorning and castration equipment, tattoo instruments, etc. According to one report, a needle used on an infected animal leads to a 60 percent chance of the next animal becoming infected when the same needle is used (J.B. Reinbold, 2010).

Finally, spread from the dam to the fetus via the placenta is possible when the dam is infected during the second and third trimesters of pregnancy. This transplacental infection could play a role in the maintenance of the disease.

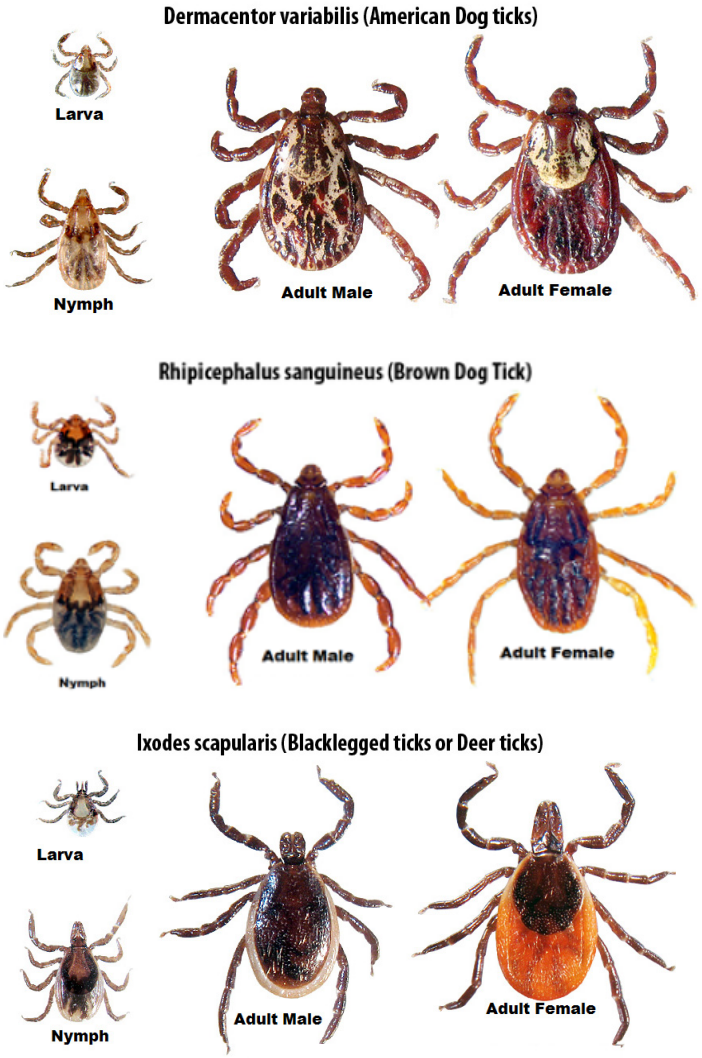

Figure 1. Adapted from tickencounter.org/tick_identification

What are the signs of anaplasmosis?

Anaplasmosis occurs in several forms: peracute, acute and chronic. Clinical signs do not typically occur until at least 50 percent of RBCs have been destroyed. Signs of acute infections include:

- Fever.
- Anorexia.
- Anemia.
- Rapid loss of body condition.
- Severe decrease in milk production.
- Pale to icteric mucous membranes (jaundice).
- Increased heart and respiratory rate.
- Muscle weakness and depression.
- Aggression and disorientation due to cerebral anoxia (especially in beef cattle).
- Abortion in females and temporary infertility in males.
- Death.
- Because hemolysis is extravascular, hemoglobinuria does not occur.

Acute anaplasmosis is most common and occurs in the summer and fall during peak vector season. When outbreaks occur at other times, iatrogenic transmission should be suspected.

In peracute infections, death occurs within hours of the onset of clinical signs. This is most common in highly susceptible animals such as purebred animals and high-producing dairy cows. These animals die so quickly that common signs such as jaundice may not be seen.

Chronic infections occur in animals that survive the initial disease. Recovery may take weeks to

months during which time production losses can be significant (decreased calf weaning weights, infertility). Cattle that have hematocrit levels below 11 have a poor prognosis. They may live, but severe hypoxia can damage the heart and other organs, leading to a chronic "poor doer." In mild cases, abortion and decreased milk production may be the only problems, which may go unnoticed.

Lesions found at necropsy include marked anemia and jaundice. The blood is thin and watery and does not clot well. The spleen is congested and enlarged with soft pulp. The liver may be enlarged and icteric (yellow) with distended bile ducts leading to a distended gall bladder. There is fluid in the body cavities and hemorrhage on the heart surface. These lesions are not enough to confirm anaplasmosis. The definitive diagnosis is made by identifying the organism on an organ or blood smear.

How is anaplasmosis diagnosed?

The most common means of diagnosing anaplasmosis in clinically affected animals is by identification of the organism on a blood smear using Giemsa stain. The organism is found in the red blood cell on the outer margins. This test can be performed both antemortem and postmortem.

For diagnosis of late clinical disease, chronic disease or carrier animals, serologic testing is performed. In the past, a card agglutination and a complement fixation test was used but is now considered less reliable due to variable sensitivity. A new competitive ELISA (cELISA) has shown good sensitivity when screening for carrier animals. The sensitivity of these serologic tests are dependent on the stage of disease and may be negative in early infections.

How is anaplasmosis treated?

Clinical signs are associated with at least 50 percent of the RBCs being destroyed while many of the remaining cells are already infected. Because of this, changing the course of the disease is difficult, even with treatment. The treatment of choice is currently tetracycline antibiotics. Prompt administration is key to survival when given early in acute disease. Many times, cattle are not recognized as infected until they are severely anemic, and treatment may not help. In some cases, the stress of handling the animal is enough to kill them, especially in beef cattle. Treatment of acute disease consists of two IM doses of long-acting oxytetracycline at 20 mg/kg at 72-hour intervals. Blood transfusions may be necessary to restore packed cell volumes and increase survival rates in more severely affected animals.

There is some debate about the possibility of clearing the carrier state in cattle. Older studies showed the carrier state could be eliminated using a long-acting oxytetracycline for three to four doses within one week used at 20 mg/kg dose IM. However, new studies have shown that not to be the case. These newer studies used more sensitive testing to identify the organism and found that animals that were infected were not able to be cleared of the disease. For this reason, cattle that are once infected should always be considered infected, even if they have been treated according to the older practices. For cattle in endemic areas, the mortality rate is rarely high, but the subclinical disease (abortions and decreased milk production) may warrant treatment. For herd in non-endemic areas, mortality rates are higher. Therefore, once an animal has been diagnosed with clinical disease in these areas, treatment of the entire exposed group should be considered.

How is anaplasmosis prevented?

In non-endemic areas, the goal is to prevent anaplasmosis from entering into the herd. Any new additions should be screened with the cELISA or purchased from a proven negative herd. Because no test is perfect, the cELISA could allow for disease positive animals to test negative. The potential wildlife reservoirs may also allow for introduction into a naive herd. For this reason, use an individual sterile needle for all animals and wash and disinfect equipment between handling animals. Control of the arthropod vector may be a viable option for some, as well as decreasing fly numbers that may act as a mechanical vector.

When available, vaccination should be considered, especially in valuable animals. A vaccine from University Products is currently available for use in some states, including Tennessee, as a USDA experimental vaccine. This vaccine produces immunity from multiple doses of killed bacteria within the vaccine. This does not prevent infection but lessens the severity of the disease. More information on the current vaccine is available at anaplasmosis.com.

For infected herds in endemic and non-endemic areas, the goal is to minimize losses. With the

potential for transplacental infections, vector transmission, wildlife reservoirs, and potential for false negative on serologic testing, it is difficult to eliminate the disease once a herd is infected. For infected herds in non-endemic areas, continuously screen for carrier animals and remove through periodic testing with the cELISA. Change needles diligently and disinfect equipment to prevent further spread. Vaccinations or feeding chlortetracycline in feed or mineral supplements during the vector season may also help. However, intake of these products is variable from animal to animal leaving some animals vulnerable. These measures will not prevent animals from becoming carriers, but will decrease chances of abortions, milk production losses and clinical disease. Supplements containing tetracycline must be labeled for anaplasmosis prevention and require a VFD from a veterinarian.

For cattle in endemic areas, the risk of disease introduction is continual. Because carriers are more resistant to disease, it is not recommended that herds in endemic areas remove carrier cattle. Control of transmission is concentrated on older, more susceptible animals because young animals are more resistant to disease. A new needle should be used for each pregnant replacement heifer and any animal 2 years of age and older to prevent spread to animals most likely to abort or have acute infections. Chlortetracycline in feed or mineral supplements may also be helpful. Again, intake of these products is variable from animal to animal. With the high exposure pressure in endemic areas, some animals may still be vulnerable. If available, providing immunity through vaccinations is the most effective means of preventing losses in herds in endemic areas. Vector control and good biosecurity practices are important in decreasing the spread of anaplasmosis in all herds.

Introducing cattle from non-endemic areas to endemic areas should be done carefully. If possible, introduce new animals during the non-vector season (if there is one). Vaccination on arrival is recommended. If vaccination is not available, consider treatment of the new animals with long-acting oxytetracycline two weeks after arrival if introduced during the vector season.

Conclusion

In conclusion, bovine anaplasmosis can be a devastating disease for your cattle herd, and it costs the U.S. cattle industry millions of dollars each year. There is still much that is not understood about the disease, including the relative importance of wildlife reservoirs. However, with the availability of more modern research techniques, these questions can start to be answered. Hopefully in the near future, we will gain a better understanding of the epidemiology of this disease and have better methods of diagnosing, treating and controlling it.

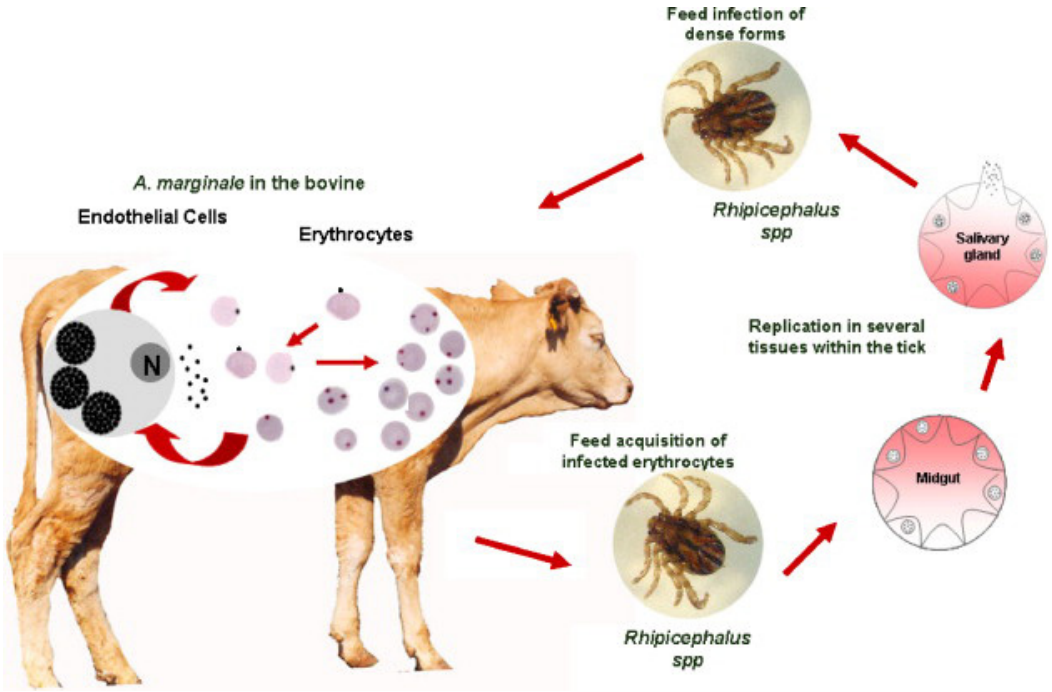

Figure 2. Molecular epidemiology of bovine anaplasmosis with a particular focus in Mexico. Rodriguez 2009

References

(2015). *Bovine anaplasmosis*. OIE Terrestrial Manual. Retrieved from http://www.oie.int/fileadmin/Home/eng/Health_standards/tahm/2.04.01_BOVINE_ANAPLASMOSIS.pdf

J.B. Reinbold, J. C. (2010, Oct). Comparison of iatrogenic transmission of Anaplasma marginale in Holstein steers via needle and needle-free injection techniques. *American Journal of Veterinary Research*, 1178-1188. doi:10.2460/ajvr.71.10.1178

K. Pompo, S. M. (2016). Survey of ticks collected from Tennessee cattle and their pastures for Anaplasma and Ehrlichia species. *Journal of Parasitology*.

Lew-Tabor, A. (2018). *Merck Veterinary Manual*. Retrieved from Anaplasmosis: http://www.merckvetmanual.com/circulatory-system/blood-parasites/anaplasmosis

Veterinary Medical Encclopedia. (n.d.). Retrieved from Anaplasmosis: antemortum and postmortum findings.

Whitlock, B. J. (2014). Seroprevalence of bovine anaplasmosis in the southern US. *The AABP Proceedings*. Retrieved from http://foundation.aabp.org/research_proposal/2012_grant_Whitlock.pdf

AG.TENNESSEE.EDU

Real. Life. Solutions.™

W 781 09/18 19-0038