

INFECTIOUS DISEASES OF WHITE-TAILED DEER EPIZOOTIC HEMORRHAGIC DISEASE (EHD)

Joe A. Ables, D.V.M, M.A. Decatur, Texas

We have all have personally experienced the impact of Epizootic Hemorrhagic Disease (EHD) for the past 10-15 yrs. But what have we learned new about this virus? What are the dynamics of EHD and how does it affect the body? What separates this virus from other viruses? Why are these viruses so problematic now than ever before? Why are vaccines not the only tool we need in our arsenal to fight with? Can we expect to see this problem eradicated in the near future? There are many questions that have been asked and in this article we will shed light on as many as possible.

What is new about EHD this year?

Currently, there are 8 serotypes, or strains, of EHD in the USA. We commonly deal with EHD 1 and EHD 2. We have seen an increase in EHD 6 which can be as devastating as EHD 1. After it is transmitted by its vector (culicoides), the EHD virus enters the blood stream and localizes within the circulatory system including the spleen where the virus resides within the membrane of the red blood cell (RBC). Unlike other viruses, EHD does not alter the membrane of the RBC thus preventing the spleen from recognizing all of these infected cells. Thus, not all the infected cells are removed from circulation. The spleen's job is to act as a check station for foreign invaders in the circulatory system and when these "infected cells" bypass the spleen, the virus has an increased time period to replicate themselves within the membrane of the RBCs. The virus produces viral particles and releases these devastating particles into the bloodstream. A viremia is simply stated as "a virus in the blood stream." The larger vessels in the circulatory system are not as affected as severely as the smaller vessels and capillaries initially due to their surface area. Capillaries are the smallest blood vessels and they are found in the extremities and major organs by which arteriolar blood delivers oxygen to the tissues/organs for cellular nutrition and function. Therefore, since the vessels are smaller in the periphery of the body, they sustain the most severe damage. The severity and type of clinical signs we observe depends upon where in the virus first affects the body. This is why there is such a variety in clinical signs. The earliest physiological signs are "leaky" vessels, kidney failure (due to decreased oxygen delivery to this highly vascularized organ) and fever. Clinically, we observe many early signs including death, laminitis (founder)-separation of the hoof wall from the toes causing "walking on egg shells." Obviously, EHD causes acute deaths when it affects the most critical areas of the body first. When the virus first attacks a less critical area of the body, the body is allowed a brief time period to fight which allows us to detect early signs of EHD infection such as swollen heads and throats. Increased salivation is usually noted early on because of increased swelling of the tongue. We see increased salivation because the tongue is swollen enough to prevent swallowing.

A typical virus is detected by the body's immune system and elicits an immune response. Therefore, the advanced avoidance of the defense system, demonstrated by EHD, elicits a postulation that deer will harbor the EHD virus in the body for extended periods of time, thus delaying the body's immune response. Extreme environmental stressors will decrease the effectiveness of the deer's immune system and allow the EHD virus to gain a stronger foot hold against the deer's defenses. One of the largest environmental stressors we observed in the year 2011 was a severe drought

and extreme temperatures in Texas. Infected deer that survived the first few hours of the EHD virus had fevers greater than 106 degrees combined with ambient temperatures exceeding 110 degrees as well as ground temperatures above 110 degrees, in the shade. This led to the wide spread demise of deer in Texas. For example, a deer with an EHD viremia has a self induced fever to naturally combat the infection in the bloodstream compounded with “leaky” blood vessels and decreased oxygen to critical organs causing a perfect setup for disaster. The drought obviously decreased the number of water available for wild deer to drink from causing increased concentration of deer in an area especially at watering holes. Meanwhile, another devastating force had been replicating in these water holes, culicoides. Increased concentrations of deer and increased exposure to culicoides gnats also attributed to the disaster. We have also experienced elevated temperatures across the United States even for extended periods of time especially in the northern states. Northern states are beginning to experience more and more cases of EHD due to possible migration of culicoides to new regions which is devastating to regions that have not had prior exposure to EHD.

How Do We Combat EHD?

Early detection is the most critical component for a treatment plan for an EHD outbreak. Fortunately, we have a few keys to help us prepare. One of the most obvious clues for detection of an EHD outbreak is the time of year. We have observed a few abnormal cases as early as April of each year but a majority of the time we expect the outbreak to begin towards the end of the summer. Knowing how the virus functions and how it attacks the body is critical. Knowing your deer herd and being in the pens daily with them is crucial. Early detection, isolation, and quick planning are critical. If a deer is suspected of having EHD and treated, even if it does not have EHD, the first level of treatment will not be detrimental to life other than a possibility of injury while isolating.

DO NOT ANESTHETIZE THESE DEER BY CHEMICAL IMMOBILIZATION.

We have studied that one of the earliest signs that we cannot visually see is “leaky” vessels. Dexamethasone is a steroid that will decrease fever/inflammation and help maintain the integrity of the blood vessel walls. The critical first level of treatment and first drug of choice to battle EHD would therefore be Dexamethasone. But as with all steroids, remember that they decrease the body’s immune system as well so be sure to get an excellent broad spectrum antibiotic on board quickly. One of the best families of antibiotics to have available is the Cephalosporins. I like Excede because of its long duration of action when given under the skin. What have we learned from this year that we can add or remove from our previous cook book recipe treatments for EHD? Another physiologic effect that EHD invokes on the body is dehydration and erosion of mucosal surface membranes, i.e. damage to the intestinal mucosal lining. Banamine is a non-steroidal anti-inflammatory drug (NSAID) that can exacerbate the effects of EHD when an animal is compromised to dehydration or intestinal mucosal injury. Banamine can impair kidney function in a deer that is already dehydrated by further decreasing blood flow to the kidneys causing irreversible damage to the kidneys. NSAIDs have been reported to cause ulceration in the intestinal lining, leading to further blood loss as well as protein loss. The older treatment routine for EHD consisting of Banamine, Dexamethasone, and Excede should be altered in your plan.

Remember, not all deer are affected at the same part of the body at the same time even though they show similar signs. Deer that have the Gastro-Intestinal (GI) component of the virus should not receive Banamine because it could possibly further ulcerate the intestines and create a larger problem. Therefore, if faced with an acute outbreak of EHD, simply removing Banamine from

your current protocol could save on treatments and treatment duration. One caveat, pregnant does should not receive steroids- Dexamethasone. Therefore, only in this situation can we use Banamine. If Banamine is the only choice we have, REMEMBER, and treat at 0.25 cc per 100 lbs in the muscle. This low dosage will accomplish what you desire and will have less effect on the GI lining. Unfortunately, it can be difficult to have your local Veterinarian on the farm within hours of an outbreak and time is critical when facing the onset of EHD. For emergency situations and if unable to reach your Veterinarian immediately, start the first level of treatment and do not forget to consult with your Veterinarian after this treatment. Your Veterinarian has an entire assortment of diagnostic capabilities that includes examining the blood and organs which is critical in implementing a direct treatment plan for your deer. EHD serotypes and titers can be obtain at this first level treatment and sent off to the lab which is important information the Veterinarian needs to attack the disease process immediately thus allowing greater success at survival.

In the fall of 2011, a private study by a Veterinarian was conducted and a treatment for EHD was discovered. The study involved four ranches totaling 81 deer with clinical signs and diagnosed with EHD 1 and 2 from the Texas Veterinary Medical Diagnostic Laboratory (TVMDL) in College Station, Texas. The research also completed the first available Serum Neutralization (SN) testing for EHD at TVMDL. What is the importance of this study? As an industry, we now have the ability not only to detect which serotype we are dealing with more accurately but also the evaluation of the “amount” of EHD in the deer. This allows Veterinarians more specific treatment options to battle specific EHD serotypes. Thus, EHD can be treated with an immune product specifically designed for EHD 1, 2, or 6, or a combination of the serotypes. TVMDL currently has the ability to detect the level of EHD in the deer for types 1, 2, and 6. The results from the research study concluded a survival rate of over 83.75% in deer with clinical signs of EHD and clinically diagnosed with EHD. EHD 1, 2, and 6 have also been isolated, which is critical for the maintenance of SN testing and for further research. Our industry now has the ability to research the deer’s immune response to EHD more effectively and we have a treatment.

With the treatment in hand, a Veterinarian can now neutralize the EHD viremia in infected deer thus preventing further viral particles from showering the circulatory system. This is not the end of the treatment plan for the affected deer though. Veterinarians and breeders must work together to treat the damage that the virus has begun to create already. Many infections, such as pneumonia and GI ulceration, may not show clinical signs for a few days after treatment of EHD. Combating the viremia quickly is critical to prevent damage to other tissues and areas of the body. Remember, preventing viral shedding into the bloodstream is critical to prevent secondary and tertiary infections from developing thus increasing the deer’s prognosis. Contact you Veterinarian to develop a Herd Health Plan to prevent EHD and have a game plan ready in case you encounter EHD in 2012.

With so much variability with this virus, will there ever be a time when we can speak of EHD as a disease of the past? With continued advances in EHD research, biochemical blood analysis, and implementation of individual treatment plans, I truly believe we can make EHD a disease of the past. We need to focus more attention than ever before on EHD and increase research even if that means spending more money. This disease has had the largest impact in our industry, and we need to be serious and not loose focus on what we have learned. Viruses are very tricky to work with, and once we think we have them figured out, they can mutate.

The year 2011 will go down in history as one of the worst years we have experienced with EHD in this industry but at the same time it was one of the biggest blessings because of the enormous amount of information we gathered at the same time. There have been diagnosed combined strains within the same herd (ranches have demonstrated to have concurrent EHD 1 and EHD 2 outbreaks at the same time) as well as outbreaks of EHD 2 followed up by EHD 1 within weeks on the same ranch. For the first time, we have observed deer to test positive to EHD 1, 2, and 6 concurrently on the same ranch as well. One goal of our society should be to combat and control this virus in every attempt to preserve our species we share in order to improve the overall health, immunity, and knowledge to fight EHD and to make it a disease of the past.

It is important to remember that antibiotics do not treat viral infections but are used to combat secondary infections left behind from the virus. It is also critical to know the process of a disease in order to specifically target it with a concise treatment plan. Viruses weaken and deplete the body's immune system allowing for increased susceptibility to common bacteria already located within the body. Early detection is step one. A Herd Health Plan must be comprehensive. Simply relying on a particular supplement, vaccine, antibiotic, steroid, or a treatment for EHD alone will not provide continued success. Incorporate all parts of your plan with your Veterinarian. Remember to develop your plan before a crisis chooses your plan. If you knew you were going to be exposed to an illness in the months to come, what would you do to prevent yourself from getting sick?

God bless and good luck!!!

Joe Ables, D.V.M, M.A.

drjoeables@yahoo.com