



December 2006

Previous VET NOTES

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- September 2006 - Gastroscopy
- August 2006 - Rhodococcal pneumonia
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Neonatal Isoerythrolysis in foals—recognition, treatment, and prevention

Neonatal Isoerythrolysis (NI) is a life threatening cause of anemia in neonatal foals. The term means that the foal's own red blood cells (RBCs) are being destroyed (lysed) by antibodies from another horse (the mare). As a consequence of the red blood cell destruction, hemoglobin is released. Once so much hemoglobin is released that it overwhelms its own metabolism, bilirubin accumulates in the blood. A yellow color appears on the skin, mucous membranes, and sclera, which is normally the white part of the eye. The foal is then said to be jaundiced which is where a lay term for this condition arises: jaundiced foal syndrome. Another medical term for jaundice is icterus. The destruction of red blood cells can cause anemia so severe that it results in the death of the foal.

Foals affected with NI have their red blood cells destroyed by antibodies they have ingested from the dam's colostrums. Usually colostrum contains only “good” antibodies that give the foal its first protection from various diseases. However, some mares have made antibodies against certain red blood cell types that they do not have. For this to have occurred, the mare's immune system has been exposed to a red blood cell type that she does not have. This exposure could have been via a previous pregnancy where the foal inherited the blood type from the stallion OR from a blood transfusion. Exposure during pregnancy is thought to occur from leakage of the foals red blood cells across an inflamed placenta or at the time of the delivery. If the mare has a subsequent pregnancy with the foal inheriting this same foreign blood type, she will respond with antibodies which will be incorporated into her colostrum. Once ingested by the foal, the antibodies cause clumping and lysing of the red blood cells - a hemolytic anemia.

Recognition and Diagnosis

Affected foals are born normally and must have had good colostrum intake. Foals can compensate incredibly well up to a point with anemia; depending on

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Carol K. Clark, DVM, Diplomate, ACVIM

the nature and amount of antibodies ingested, signs begin 8 hours to 5 days post birth. Without stress, the signs can be very subtle and include just mild inactivity and lethargy. Eventually, increased respiratory rates, elevated heart rates, weakness, lack of nursing and pale/icteric gums and sclera are seen. Fever may also occur due to the immune destruction or due to secondary infection. Pink, red or orange urine can be noted. Severe cases can seizure or die suddenly.

A complete blood count (CBC) will assess the anemia and serum chemistry results may reveal problems with other organ systems due to the hemolytic anemia. The kidneys can be insulted by the red blood cell breakdown products, mainly bilirubin, and the liver may have elevated enzymes from the poor perfusion associated with the anemia. Abnormal heart rhythms can occur from poor blood oxygenation due to the anemia and can lead to the demise of the foal. Strong evidence of the disease can be demonstrated using the Jaundiced Foal Agglutination test which tests for an incompatibility between the mare's serum and the foal's red blood cells. The definitive test for confirmation of the cause of the anemia is to test the mare's serum for the presence of anti-RBC antibodies.

Treatment

If the clinical signs are acute and severe, much caution in handling the foal is advised. Severely anemic animals can die if subjected to stressful restraint and transport. Oxygen administration can help maximize the RBC function that remains. Non steroidal and steroidal anti-inflammatory drugs are indicated to decrease the immune response. Antibiotics are also indicated to prevent and treat secondary infection that may occur from a stressed immune system. Blood may be given from a gelding, known universal donor, or washed RBCs from the mare. Our hospital uses an universal blood donor that has been tested free of anti-RBC antibodies. Milder cases with no severe signs of anemia may not need a transfusion.

The prognosis depends on how severe and how early the foal was affected. Generally, the younger the foal is at the onset of the signs, the worse the prognosis. However, the disease is generally curable if diagnosed before severe signs begin. Complications that can occur include the following: septicemia or secondary infection from a "taxed" immune system, kidney disease from bilirubin toxicity and kernicterus. Septicemia can lead to diarrhea, a/or septic joints. Kernicterus is a neurologic condition causing seizures and retardation that can occur if the bilirubin levels stay greatly elevated (>20mg/dl).

Prevention

This disease is **COMPLETELY PREVENTABLE** with proper management and testing. The goals of testing is to identify those mares at risk for having an NI foal and then screen those mares a/or their foals for incompatibility prior to the foal consuming colostrum.

There are 8 blood groups of horses and each group has various factors associated with it. The blood groups are A, C, D, K, P, Q, T, and U. The factors are denoted by lower case letters such that a blood type is expressed as a Capital letter and a lower case letter (i.e., Qa). Most reported cases of NI have occurred in response to the Qa or Aa antigens. Pa, Ab, Qrs, Dc, Ua, Qb, Qc, Da, Ka, and Db have rarely been reported to cause disease. Anti-Ca may actually be protective because Ca, Aa negative mares have spontaneously produced Anti-Ca antibody which has prevented the production of Anti-Aa antibody by removing fetal RBCs with the anti-Ca antibody before the mare can cause production of Anti-Aa antibody.

Tests before pregnancy – The most common red blood cell types associated with causing the disease are Aa and Qa which are very prevalent in Thoroughbred and Standardbred horses. Therefore, a mare that lacks one of these blood types is at risk for developing NI antibodies with enough pregnancies because so many of the stallions are likely to pass one of the blood types to his foal. It is estimated that 18% of mares from these breeds are susceptible, yet there is only a 1% prevalence reported in Thoroughbred foals. Therefore, one can test to see if a mare is even at risk to produce an NI foal by simply making sure she has these common blood types. If she has the blood type, she won't make antibodies against her own blood type.

Tests during pregnancy – High risk mares do not contain the common RBC types and should be tested during the last 2 to 4 weeks of pregnancy for anti-RBC antibody in their serum. If positive for these antibodies, foaling must be observed. The foal is then muzzled to prevent ingestion of the dam's colostrum and is fed banked colostrum from other mares. Usually 1 quart of good quality colostrum is enough to provide adequate IgG. The foal is then fed, every 1 to 2 hours, milk from another mare for 36 hours. Meanwhile, the dam is milked to remove the dangerous colostrum. Although this is labor intensive for the first 36 hours, it does allow the mare to raise her own foal after that and avoid a life threatening condition.

Tests after parturition – If the mare is at risk for producing an NI foal or has been confirmed to have anti-RBC antibody in her blood, testing for compatibility between the foal's RBCs and the mare's serum is recommended. This is called the Jaundiced Foal Agglutination Test. In this test, the foal's RBCs are mixed with the mare's blood and evidence of agglutinating (or clumping) confirms anti-RBC antibody is present. If the test is positive, the foal is not allowed to nurse its mare for 36 hours and an alternative source of colostrum and milk is supplied via a bottle. Meanwhile, the mare's udder is regularly stripped to remove the colostrum containing the antibodies. After 36 hours, the foal is allowed to nurse the mare normally as its gut is now "closed" from absorbing antibodies and the amount in regular milk is much lower than the colostrum.



The Third Annual Peterson & Smith Horseman's Seminar took place on Saturday and Sunday, November 18 & 19, 2006 at the Ocala Hilton. It was a fun filled factual seminar for horse owners of all breeds of horses - from miniature horses to Thoroughbred race horses.

The two-day seminar was filled with speakers from Peterson & Smith and renowned guest speakers including: Dr. Ray Geor, Nutritionist from Virginia Tech, Dr. Bryan Fraley, Podiatrist from Rood and Riddle, Kentucky, Dr. Dennis Brooks, Ophthalmologist from the University of Florida, and Dr. Rob Holland a vaccine expert from Pfizer Equine.

Topics ranged from hoof care and new concepts in shoeing, to embryo transfer, to surgical options, to vaccinations and why it is so important make sure your horses are up-to-date.

The seminar included a sponsor exhibit where the vendors were able to talk to participants about their products and provide them with samples and information. The sponsors ranged from nationally recognized corporations from across Florida to local equine businesses - including Jerry Parks Equine Insurance, Seminole Feed, and Tack Shack of Ocala.

Attendees were also able to participate in a "hands on" lab, which offered an opportunity for the novice horse owner to learn basic information such as equine anatomy, basic horse restraint, how to put on a bandage and where to give simple vaccinations.

The event was hosted at the Hilton Hotel, who catered breakfast and lunch each day and also provided an outstanding dinner on Saturday evening for the annual Peterson & Smith dinner panel. This humorous and educational panel consists of six veterinarians including the guest speakers who spend the evening answering questions from the participants. A lot of fun is had by all. The questions raised and the topics suggested by the participants will form the agenda for next year's seminar on November 17 & 18, 2007. Please mark it on your calendars and come and join us next year.

- August 2005 - Extracorporeal shockwave therapy (ESWT)
- July 2005 - Vaccination recommendations
- June 2005 - The advantages of high fat/low carbohydrate diets for horses
- May 2005 - The Hoof: Form and function
- March 2005 - Liquid gold
- February 2005 - Breeding the problem mare
- January 2005 - Condylar fractures
- December 2004 - Early diagnosis and treatment of high-risk pregnancy in the mare
- November 2004 - Know your horse
- October 2004 - White Line Disease

We're on the web:
www.petersonsmith.com



4747 SW 60th Avenue
Ocala, FL 34474

Phone: (352) 237-6151
Fax: (352) 237-0629
Email: PSEH@petersonsmith.com

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