ANAPLASMOSIS

Anaplasmosis is a tick-borne rickettsial disease caused by Anaplasma marginale. The disease is endemic in many parts of the world including the US. Prevalence is higher in the southern US and is thought to be lower in the northern US and Canada; however, anaplasmosis is still being reported for all of these areas often in proximity to river areas especially as cattle begin to travel.

Transmission is typically through ticks of Dermacentor spp. and mechanical through transmission by needles and/or surgical instruments. Biting insects are also involved in transmission.

Cattle of all ages can be infected with anaplasmosis with severity of clinical disease related to the age of the animal. Mortality is most common in animals greater than two years old. Clinical disease is often reported late August through frost.

Prevention

In the clinical setting diagnosis on a stained blood smear using Diff-Quik, Giemsa, or Wright’s stains for organisms will provide a rapid diagnosis.

Lesions

Lesions normally associated with Anaplasmosis include:

- Pale mucous membranes
- Marked icterus (yellow to golden hues of mucous membranes, conjunctiva, and areas of unhaired skin)
- Thin, watery blood
- PCV can and usually will be extremely low
- Enlarged, purplish to mushy spleen, with prominent follicles
- Hepatomegaly with yellow-orange discoloration and rounding of edges

Diagnosis

Whole blood collected in EDTA or heparin tubes from clinically affected animals is the diagnostic sample of choice. However, the ISU VDL has an Anaplasma spp. PCR available with valid sample types: EDTA blood, heparinized blood, or spleen. Additionally, a concurrent submission of air-dried, unstained blood smear preparations is also encouraged. Serology can be used to evaluate for possible infection or used on a herd basis to evaluate prevalence.

Collection of formalin-fixed and fresh tissue for differential diagnosis is preferred in most instances, particularly in areas where endemic infection is uncommon.

Vaccination does work to prevent acute expression of disease; however, it does not prevent infection or the development of carrier animals. Nutrition and environmental stress are important factors in suspect herds. Additionally, reducing vector transmission (biting insects) and mechanical transmission of instruments used to work cattle are important.

Steps of Anaplasmosis

1. Incubation stage – begins at infection. Average incubation stages are 3-8 weeks, but wide ranges have been seen. Animals appear clinically normal at this stage.
2. Developmental stage – lasts 4-9 days, characteristic clinical signs occur. 1. Forced movement and excitement can result in death due to anoxia 2. Antibiotic treatments do little to affect the outcome of disease during late developmental and early convalescent stages. Death typically occurs during the late developmental or early convalescent stages.
3. Convalescent Stage – cattle that survive clinical disease lose weight, abort calves, and recover slowly over a 2-3 month period. Increased erythropoiesis is typically observed in this time frame.
4. Carrier Stage – unless medicated appropriately cattle that recover from anaplasmosis remain carriers for life. During the carrier stage, animals do not exhibit clinical signs.

Stages of Anaplasmosis

1. < 6 months old
   - Rare, but can become infected
2. 6 – 12 months old
   - Mild subclinical disease
3. 1 – 2 years old
   - Acute but rarely fatal disease
4. 2 years old
   - Acute and often fatal disease
5. If older than 2 years
   - Clinical signs present initially with anorexia, off feed, constipated with pale mucous membranes. Dyspnea and exercise intolerance may become apparent, with marked aggregation common. More often present as “sudden death”. After 3 years of age, 30-50% of clinical anaplasmosis cattle die if untreated.

Lesions

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Erin grew up in Bettendorf, Iowa and attended Iowa State University. She graduated with a Bachelor’s Degree in Biology in 2005.

Erin started working at the ISU VDL in 2006 running IFA and ELISA tests in the Serology section. She continues to run Serology tests, but also serves as a Quality liaison and is the VDL’s Environmental Health & Safety contact.

Erin met her husband Gary while attending ISU. She was in the marching band and pep band and currently is the President of the ISU Alumni Band. Gary was on the mascot squad, so the two enjoy attending ISU sporting events including football, basketball, volleyball, and gymnastics. They have a beagle, a rabbit, and also foster animals for local rescues.

**Question:**

How are these 3 sets of results possible?

- **Serum PCR + and ELISA –**
- **Serum PCR + and ELISA +**
- **Serum PCR – and ELISA +**

**Answer:**

It depends where the pigs were located on the “PRRSV infection curve” when they were sampled. (See Fig. 1)

**Serum PCR + and ELISA –** happens very early, normally within the first few days, after exposure in the course of PRRSV infection. Pigs are viremic but have not seroconverted yet (acute phase).

**Serum PCR + and ELISA +** happens from around week 1 until about week 5, after exposure in the course of PRRSV infection. The pigs are still viremic and are starting to seroconvert (transition from Acute to Chronic phase).

**Serum PCR – and ELISA +** happens from week 6 after exposure until several months after virus has been cleared from tissues. The pigs are no longer viremic and most animals have seroconverted and show high levels of IgG (Chronic phase).

The importance of antibodies and their role in protection against PRRSV has been unknown until recently. Specifically, the role of neutralizing antibody is now considered an important piece of the puzzle for protective immunity against PRRS virus. Several studies show that neutralizing antibody prevents the appearance of or blocks PRRS viremia.

PRRSV antibody responses can be detected by ELISA within 7 to 9 days post infection (PI). However, there is no evidence that suggests this early response plays a critical role in protection against PRRS. This is likely due to the fact that these early antibody responses do not neutralize PRRSV in vitro, and in experimental studies, these antibodies have not mediated passive protection against pigs challenged with virulent PRRS virus. However, antibodies that do have neutralizing activity appear later at approximately 4 or more weeks post infection.

For the full paper: Role of Neutralizing Antibodies in PRRSV Protective Immunity by O.J. Lopez and F.A. Osorio, please visit our website at www.cvm.iastate.edu/vdl

**Figure 1. Temporal sequence of events after infection of a pig with porcine reproductive and respiratory syndrome virus.** (OJ Lopez and FA Osorio Role of neutralizing antibodies in PRRSV protective immunity.)

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**STAFF HIGHLIGHT**

Erin Kalkwarf

Serology

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**EZ-Ship Program**

The ISU VDL has launched an EZ-Ship program which allows ISU VDL clients to generate and utilize printed UPS shipping labels in order to ship packages to the ISU VDL from anywhere in the contiguous United States.

These single use labels can be generated and delivered electronically via PDF file from the ISU VDL Client Web Portal. They will be pre-filled with your return address and the ISU VDL shipping address. You will not be charged by the ISU VDL until the package is picked up by UPS for delivery. The shipping charges will be placed on the accession invoice (if more than 1 accession in box then charges will be split evenly).

**For more details on pricing please visit**
www.cvm.iastate.edu/vdl

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**ANNOUNCEMENTS:**

**Upcoming University Holidays:**

- **Thanksgiving**— Thursday, November 22nd
- **Friday, November 23rd**
- **Monday, December 24th**
- **Tuesday, December 25th**

**HATS will be closed on Christmas Eve and Christmas Day (December 24th and 25th) but will be testing as normal on Saturday December 22nd.”**

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**Helpful Hint **

When filling out a necropsy web submission, the number of lines in the “Animal ID” table need to match the number provided in the “Number of Samples” field.