Anaplasmosis in Cattle

by
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CASE REPORT

On September 6, 1975, one cow in a herd of 36 near Aplington, Iowa, was found by the owner as being slow and weak. Dr. Eldon Uhlenhopp was called to examine the cow. The cow's mucous membranes were found to be very pale and icteric. She also had a temperature of 100°F. Hemoglobin was determined to be 3 gm %. A differential diagnosis of anaplasmosis or leptospirosis was made. The cow was treated with 500 mg. oxytetracycline and 20 cc Vitamin B-complex intravenously, 10 cc iron dextran and 10 cc methioplex intramuscular. Serum samples were sent to the Veterinary Diagnostic Lab for anaplasmosis and leptospirosis serological examination.

On September 11, 1975, the cow examined and treated earlier died, and a second cow was showing similar clinical signs. She was treated with the same regime as the first cow. On September 14 a third cow was found sick. The second and third cow were then both treated. In spite of treatment, both cows died on September 16.

On September 18, the Veterinary Diagnostic Lab reported a positive reactor for anaplasmosis using the card test and serum from the first cow noticed sick.

The owner decided to send the whole herd to a slaughterhouse. No further deaths were reported prior to shipment.

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SUMMARY

Anaplasmosis is an infectious, noncontagious, hemolytic disease of cattle caused by *Anaplasma marginale*. It is a widespread disease that costs producers 100 million dollars annually. *A. marginale* is transmitted by arthropod vectors and animal health instruments. Infected cattle are weak and icteric. Hemoglobinuria is absent. All animals recovering from anaplasmosis are considered carriers. Diagnosis is confirmed by blood smears and serological testing. Tetracyclines are recommended for treating the clinically ill and carrier animals. Vaccination of cattle and control of transmission reduces the incidence of anaplasmosis.

ANAPLASMOSIS IN CATTLE

Anaplasma marginale invades erythrocytes causing an extravascular hemolytic condition. The severity of the condition depends upon an animal's susceptibility and ability to respond to a hemolytic crisis. Factors modifying susceptibility are undetermined in cattle native to endemic areas. In general, severe clinical infections do not occur until an animal is about eighteen months or older. Young animals become infected but rarely show clinical signs. Their recovery usually results in a carrier state (7).

Anaplasmosis is one of the most costly diseases of the beef industry. Some 50,000 to 100,000 animals die of anaplasmosis annually (6). Death loss where disease is introduced into a herd will average 10 percent and may reach 50 percent (14).

Death and morbidity losses cost our animal industry approximately 100 million dollars annually (6).

Anaplasmosis has been a disease problem primarily in the South Atlantic and Gulf Coast States. Subsequently, anaplasmosis has become common in the Northwest and California (14). In recent years, it appears to be an increasing problem in the Midwest. The Iowa Veterinary Diagnostic Laboratory, Ames, reported 223 reactors in a seven month period from the end of 1974 to the middle of 1975. Hawaii and Wisconsin are the only states requiring negative test for anaplasmosis (15). Hawaii eradicated anaplasmosis in 1954 (14). Federal law does not permit the interstate transportation of infected animals. Dr. Butler, State Veterinarian of Iowa, wants all cases of anaplasmosis to be reported, but law does not require quarantine of confirmed cases.

For many years anaplasmosis was masked by, and confused with, bovine piroplasmosis (Texas cattle fever). Theiler, in 1910, recognized anaplasmosis as causing a specific syndrome. The first case confirmed as anaplasmosis in the United States was in Kansas (1923).

A. marginale has not been classified. For many years it was thought to be a protozoan. With use of the electron microscope, the internal structure became discernible. It was found that anaplasma bodies are composed of several small initial bodies. Today it is considered rickettsial in nature (12).

With the use of Wright's or Giemsa's stain, *A. marginale* appears as a basophilic, round body situated at or near the margin of an erythrocyte. It measures 0.2 to 1.0 micron in diameter. The form normally seen is round, but a filamentous form, *Paranaplasma* sp. (12), is a common form found in deer.

A. marginale reproduces by binary fission (12). During fission, the anaplasmal bodies utilize erythrocyte cell membrane phosolipids. The resultant erythrocyte is more fragile and more attractive to the reticuloendothelial system. Microfibrils on the initial bodies may aid intererythrocyte movement; the complete explanation of movement is unknown.

Transmission of *A. marginale* involves the inoculation of infected blood into a susceptible animal. Only a minute quantity of infected blood is required; the smallest infective dose is 40 infected erythrocytes. Transmission may be either natural or unnatural, but must be within five minutes (14), because anaplasmal bodies are destroyed by hemolysis and dessication. Transmission is also more likely when parasitemia is greater than two percent (7). The principal host of *A. marginale* is the bovine, although deer have been incriminated as a reservoir (14). The full significance of deer as a reservoir has not been completely evaluated.

The most efficient unnatural transmitter is man. A 16-gauge bleeding needle will carry millions of erythrocytes and place a good share of them into the jugular vein. Many other instruments have been incriminated, including dehorning saws, castration knives, nosetongs, vaccination needles and tattoing instruments. Time should always be taken to properly cleanse and sanitize equipment following their use. The use of disposable needles and syringes is a good practice in endemic areas or in treating diseased animals for anaplasmosis.

Natural transmission is by arthropod vectors. Ticks are considered biological vectors, since the anaplasmal organism can survive in them for an extended length of time. Nineteen species of ticks are reported to transmit *A. marginale*. *Dermacentor andersoni* and *Dermacentor occidentalis* are among those considered most responsible (11). The horse fly is a very efficient transmitter since their mouth parts are suitable for biting and carrying blood. Deerflies, stable flies, and mosquitoes have also been documented carriers (13). The insects involved will vary according to the area of the country.

In cattle the incubation period for anaplasmosis is from fifteen to thirty-six days. There is a high correlation between clinical symptoms and the amount of inoculum transmitted. Cardinal features of anaplasmosis are anemia, weakness, fever, and normal urine. Accompanying signs are icterus, inappetence, depression, dehydration, labored respiration, and irrational behavior.

During the incubation period, anaplasmal bodies increase in number and

the animal remains asymptomatic. Parasitized erythrocytes are destroyed in the spleen and bone marrow without release of free hemoglobin, hence no hemoglobinuria. At the time of greatest fall in erythrocyte numbers, the body temperature becomes elevated. This elevation is due to the sudden release of hemoglobin breakdown by-products in the animal's system. Temperatures may range from 104 to 108 degrees F.

The degree of anemia is related to the rate of erythrocyte loss and the bone marrow's ability to regenerate immature erythrocytes. Packed cell volumes (PCV) can range from 7 to 25 percent with hemoglobin values of 2.3 to 8.2 grams percent (gm %) (10). The degree of anemia can be roughly estimated by the degree of pallor present. The conjuctival membranes are consistently pale when 40 to 50 percent of the erythrocytes are depleted. When erythrocyte depletion is greater than 60 percent, nasal mucosa, vulva, and skin become pale (10).

Peracute cases of anaplasmosis are more common in cattle three years of age or older. In these cases, there is a sudden onset of high fever, anemia, icterus, severe dyspnea, and death within 24 hours.

Most cases are subacute, with a temperature rise for several days which stabilizes and may persist for two weeks. The PCV usually is above 11 percent. It takes approximately ten days to see an appreciable increase in PCV. The bone marrow responds by adding immature erythrocytes to the circulation. These immature cells are refractory to anaplasmal bodies. Anaplasmosis in the recovery phase is characterized by a transitory macrocytic, hypochromic anemia. An accompanying leukocytosis is consistent with an anticipated increase in granulopiesis in a hemolytic crisis. At a later stage, reticulocytosis and polychromasia would be seen in a blood smear (10).

Convalescent animals are usually icteric, due partly to the liver damage caused by anoxia during peak parasitemia and hemolysis. Convalescent animals are also predisposed to cardiac failure. Therefore, they present an additional challenge in handling for treatment. The stress of being driven to a corral may result in myocardial failure.

A tentative diagnosis can be made from clinical signs and post-mortem evaluation. History is often helpful, since an outbreak is often associated with bringing new cattle into the herd. The absence of hemoglobinuria is good supportive evidence, since hemoglobinuria is present in other diseases likely to be confused with anaplasmosis, such as leptospirosis.

Necropsied animals will show lesions associated with anemia. The blood is usually watery. The animal carcass is emaciated and dehydrated. An enlarged, mottled yellow to brown liver with a distended gall bladder is common. Splenomegaly is quite marked. Serous atrophy of renal and cardiac fat, myocardial hemorrhages, and pulmonary edema are other lesions often present but not always pronounced.

A confirmatory diagnosis is made with blood smears or serology. A blood smear stained with Wright's stain should be examined for anaplasmal bodies at the margin of red blood cells. Peripheral capillary blood is preferred, since infected cells tend to concentrate in small vessels (7). The card test, which is official, will give accurate results in ten minutes (14). It requires serum; a positive reactor will show blue-green agglutination. Other serological tests are available in diagnostic labs. They include the complement-fixation and capillary tube agglutination tests.

All clinically ill animals should be isolated from the rest of the herd. High parasitemia immensely increases the probability of vector transmission. Commercial insecticides may be advisable if complete isolation is impossible. Not only does isolation reduce chances of transmission, but it aids in recovery because a more suitable environment can be established. Fresh water, palatable feed, and less stressful treatments should be provided. Any factor removing stress is beneficial to the animal's recovery. These diseased or convalescent animals are prone to secondary infection.

Tetracyclines are recommended for treating anaplasmosis. The intramuscular or intravenous administration of oxytetracyclines (22 mg/kg) will help clinically ill animals (6). Prolonged ad-

ministration is not required. Like all infectious diseases, prognosis is best with early treatment. Supportive therapy includes hematinics, B-vitamins, and energy-enriched electrolyte solution. Blood transfusions are often indicated but usually reserved for expensive breeding cattle. The amount used is variable, but ½ to 2 gallons of citrated blood given intravenously will improve a clinically ill animal. Caution must be enforced when administering blood or fluids. Most animals are prone to cardiac embarrassment, so fluids must be given slowly. A guarded prognosis is given when total erythrocyte count is less than two million per cmm (10).

All animals recovering from anaplasmosis are considered carriers (7). The carrier can be treated either parenterally or orally with chlortetracyclines at the daily rate of 5 mg. per pound of body weight orally for 45 days, or at the same rate parenterally for 10 days (11). Serological testing of these carrier animals is recommended 6 to 8 months later. When the carrier state is eliminated, these animals are susceptible to reinfection by anaplasmosis. Two drugs, imidocarb and thiosemicarbazone, not yet approved for use in food animals, are reported effective in ridding the carrier state after one or two injections (5,8).

The inaguration of regulatory control programs by the federal government has been handicapped by the lack of money to fund slaughter indemnities, herd testing, and hiring regulatory officials. Without federal support, control of anaplasmosis is unfeasible. Today, anaplasmosis continues to spread in spite of the fact it can be controlled.

The livestock producer can do several things to protect his investment. Fort Dodge Laboratory has an inactivated vaccine, Anaplaz® , available for commercial use. The vaccine does not prevent infection but does eliminate or drastically reduce the severity of the clinical disease. The recommended dosage schedule is two injections four weeks apart, preferably given to open heifers. An annual booster is recommended. The protective titer will persist for approximately eight months. There are reported cases of neonatal isoerythrolysis in calves born to vaccinated cows. The isoantibodies are concentrated in the colostrum. The incidence is variable, but Fort Dodge Laboratory reported 1:8000.

The lievestock owner can have his herd screened with the card test, which costs approximately one dollar per head. He can either sell reactor animals for slaughter, or segregate the herd. A fence without water facilities or trees where cattle can congregate is adequate for segregation. As the reactor cows age, the owner can then cull these animals and replenish the herd with offspring from negative cows.

Control of anaplasmosis in endemic areas has been accomplished by the continuous feeding of chlortetracycline (0.5 mg/lb.) (14). It can be incorporated into salt-mineral mixes, blocked feed, and range cubes.

Regardless of the measures undertaken, control of arthropod vectors and ticks is essential. Many endemic areas can reduce anaplasmosis incidence only when they reduce the insect vector population.

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Lead Poisoning in Dogs

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SUMMARY

Lead poisoning is seen primarily in dogs less than one year old; the highest percentage of those are less than five months old. Many sources of lead are available to young dogs, although lead-based paint and linoleum are the principal problems. The characteristic clinical signs involve the gastrointestinal system (vomiting, colic and diarrhea), nervous system (hysteria, convulsions and nervousness) or both. Generally the signs are followed by a period of normalcy. Dogs are unique in that clinical pathological analysis showing many nucleated red blood cells and basophilic stippling in the absence of severe anemia is strongly suggestive for lead poisoning. However, analysis for blood lead with over 0.35 ppm. is the best diagnostic tool. Chelation therapy with Ca EDTA is the treatment of choice.

CASE REPORT

On August 1, 1975, a one-year-old male beagle was admitted to Stange Memorial Clinic as a lead poisoning suspect. The dog's history included vomiting and abdominal pain for the past 10 days and nervous signs of two days duration manifested by convulsions, running, crying and apparent blindness. The dog reportedly had been tethered adjacent to an old building from which the owner had been scraping paint. On outpatient examination, the beagle appeared depressed and emaciated and vomited a yellow foamy material. He had reportedly not eaten any significant amount of food for about three weeks. When left to himself, he would stretch out his front legs and rest his head on them while keeping the hind legs erect. Abdominal radiographs revealed numerous flecks in the colon which could have been lead-based paint chips. Blood samples for hematologic examination and lead analysis were taken. Chelation therapy was initiated on the basis of the radiographs. He was given 12 mg/lb. of Ca EDTA subcutaneously. The blood analysis showed a hemoglobin of 13.6, PCV of 42, and a corrected WBC of 8,625. There were 387 NRBC/100 WBC, along with basophilic stippling, polychromasia and anisocytosis. The blood lead level was 1.34 ppm. The dog was given several enemas initially and was continued on Ca EDTA, lactated ringer's and glucose, periodically, for 14 days. He had several convulsions and went into more severe depression at the onset of Ca EDTA

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