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Anaplasmosis in a Hereford Cow

By

R. N. Lanz* and B. L. Hull, D.V.M.†

Introduction

Anaplasmosis is a condition recognized more frequently in the bovine in recent years. However, even more important is the fact that it is becoming more prevalent in areas outside the epizootic areas. The organism was first observed by workers studying Texas cattle fever, therefore it is plausible these men were often seeing cattle with two conditions (9). Théler in 1910 was the individual who recognized the organism as causing a specific disease in South Africa and named it *Anaplasma marginale* (9). The first field case reported in the United States was described by Darlington in Kansas in 1926 (4).

The disease has been reported in all states except Alaska and the New England states, and until recently Wisconsin had not reported any cases (9,6). It is reported enzootic in the southeast, southern coastal areas, west coast and parts of Kansas, Oklahoma, Missouri, Illinois, Kentucky and Tennessee (5). The economic loss is reported to cost the livestock industry 35 million dollars annually (9).

This report describes anaplasmosis in a
Hereford cow that was admitted to the Iowa State University (ISU) Veterinary Clinic from a herd of Hereford cows in eastern Iowa.

Case Report

A three year-old Hereford female weighing approximately 800 pounds was admitted to the clinic on November 12, 1970. She was from a herd of 70 Hereford cows of similar age and weight in east central Iowa. This herd was purchased in the upper peninsula of Michigan on October 10, 1970.

The referring veterinarian reported seeing the first clinically sick animal in the herd on November 6. This animal showed signs of icterus, dehydration, anorexia, listlessness, difficulty in moving and tiring easily when driven. Two animals died on that day and by November 8 one more animal was dead and two more started showing clinical signs. By November 11, 25 cows had or were showing clinical signs and seven had died. The treatment of the sick animals consisted of tetracyclines intramuscularly (i.m.) and orally, triple sulphonamides, penicillin-streptomycin, vitamins A, D, and E and the whole herd was vaccinated with a combination of Leptospira pomona, icterohemorrhagica and canicola vaccine and was given a single injection of streptomycin. The veterinarian had tentatively diagnosed leptospirosis and/or anaplasmosis and vaccinated as described. He also contacted the state district veterinarian and took blood samples for serology. He did a post mortem examination and reported seeing severe icterus, watery blood and hemorrhages at the apex of the heart.

On November 12 the cow was brought to the ISU clinic with serum samples from her and two other clinically sick animals in the herd. The veterinarian requested serology tests for anaplasmosis and leptospirosis. The cow was not eating, had a temperature of 101.5° F., had normal appearing feces, pale mucous membranes, normal heart and lung sounds and rumen atony. Blood was taken for a complete blood count (CBC), blood parasites, serological test for anaplasmosis and urine was taken for urinalysis (Table 1 & 2). Blood was collected from a clinically normal cow in the ISU reproduction teaching herd and administered via the jugular vein. Approximately 250 cc of blood was given when the cow began to tremble, appeared weak and collapsed, 10 mg of dexamethasone was injected i.v. immediately. Within one hour the cow appeared to have returned to her previous clinical state.

The following day the clinical pathological examination was completed (Table 2). The capillary-tube agglutination test† was positive and a positive test for Leptospira pomona, icterohemorrhagica and canicola was also noted. Clinically the cow was similar to the previous day but the feces was much firmer. Four thousand mg of oxytetracycline HCl t in 500 cc physiological saline was injected i.v.

On November 13 the cow began eating some grain and the feces was still firm.

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**TABLE 1—HEMATOLOGY**

<table>
<thead>
<tr>
<th>Date</th>
<th>Sample Obtained</th>
<th>11–12–70</th>
<th>11–16–70</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hb (Gm %)</td>
<td>2.8</td>
<td>3.5</td>
<td></td>
</tr>
<tr>
<td>PCV (%)</td>
<td>9.4</td>
<td>12.5</td>
<td></td>
</tr>
<tr>
<td>RBC (10⁶)</td>
<td>(….)</td>
<td>2.83</td>
<td></td>
</tr>
<tr>
<td>WBC</td>
<td>10,450</td>
<td>19,500</td>
<td></td>
</tr>
</tbody>
</table>

**Blood Smear**

- Baso. | 0 | 0 |
- Eosino. | 0 | 0 |
- Seg. Neut. | 56 | 60 |
- Band Neut. | 6 | 7 |
- Lympho. | 37 | 33 |
- Mono. | 1 | 0 |
- Platelets adequate | adequate |

**RBC Morphology**

- 9% nucleated | RBC poly-chromasia |
- 4% nucleated | RBC poly-chromasia |
- anisocytosis |

**TABLE 2—URINALYSIS**

| Color | coffee brown |
| Sp. Gr. | 1.020 |
| Reaction pH | 6.5 |
| Albumin | 30 plus |
| Acetone | negative |
| Sugar | negative |
| Blood | negative |
| Bilirubin-Conj. | trace |
| Urobilinogen | 1 |
| sediment | vaginal epithelial cells |

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*Azium, Schering Corp.
†Anatest, Diamond Laboratories
‡Liquamycin, Pfizer Inc.
The cow was treated with 3500 mg of oxytetracycline HCl in 500 cc saline and this was repeated the following three days. By November 16 the patient's appetite appeared normal, the feces was normal in consistency, the mucous membranes appeared to have more color and a blood sample was taken for a CBC (Table 1). No treatment was given on November 18 since the cow appeared quite bright and alert, had a good appetite, normal temperature and feces.

On November 19 the cow's temperature was 103.0°F., respiration was increased, the animal was slightly depressed and there was a bilateral mucous discharge from the external nares. Treatment consisted of 25 cc of peicillin-streptomycin* administered i.m. and the cow was discharged from the clinic.

Discussion

Clinical signs—The clinical signs observed during the eight days at the ISU clinic were similar to those described by others (3, 7, 11). The most commonly described signs are anorexia, depression, increased respiration and pulse and progressive anemia with eventual icterus. The most striking things were the extreme pallor of the mucous membranes and the watery consistency of the blood. Variable temperature responses are reported but it is generally agreed that a rise in temperature will be observed in the course of the disease (3, 7, 11). Schalm (2) reports the increased temperature is due to large amounts of hemoglobin (Hb) or Hb by-products being released into the blood stream. Since the initial Hb level noted in the clinic was 2.8 Gm %, most of the Hb loss had already occurred, therefore it is possible the temperature response occurred prior to seeing the cow in the clinic. A slight temperature increase was noted on the eighth and last day in the clinic but a concurrent bilateral nasal discharge was also noted, therefore the animal may have been developing a secondary respiratory infection. Christensen (3) does report observing drooling and mucopurulent nasal discharge in uncomplicated cases of anaplasmosis.

Clinical Pathology—The laboratory results were very typical of those reported by others (3, 7, 11), and were essential in confirming the diagnosis. The two most significant abnormalities noted in the hemogram were the values obtained for Hb and packed cell volume (PCV) (Table 1). It is noted that there is a bone marrow response indicated on the first day's tests by the number of nucleated red blood cells, polychromasia and anisocytosis, therefore it is possible that the Hb and PCV may have even been lower prior to this time. Within three days after treatment both the Hb and PCV had increased and clinically the cow was improving. The marked neutrophilia probably is a response to the vast erythrocyte loss and had progressed enough to cause a leukocytosis by the fourth day in the clinic.

The urinalysis (Table 2) indicates an acid pH, probably due to rumen stasis and the proteinuria may be indicative of kidney tissue hypoxie as the result of anemia.

Confirmation of the diagnosis was made with the aid of the blood smear, which was used to demonstrate the typical bodies in the erythrocytes, and the positive CA test. The positive tests for leptospirosis may be the result of the recent vaccinations.

Treatment—As with many other diseases, workers experimented with many agents such as arsenicals, antimalarial agents and broad spectrum antibiotics before finding an effective drug (7). It was found that the most effective drugs were the tetracyclines at 3–5 mg/lb body weight. One dose may be effective but to eliminate the carrier state, 5 mg/lb of the drug must be given for 10 days parenterally or orally for 60 days at the same dosage rate (3). More recently Bedell and Salter (2) have shown that two doses of oxytetracycline at 1.5 mg/lb in combination with an anaplasmosis vaccine* was effective in reducing clinical signs and producing immunity during a heard outbreak. In addition to elimination of the organism, treatment should be directed

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* Combiotic, Pfizer Inc.

* Anaplaz, Fort Dodge Laboratories

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toward reducing the anemia also. It is recommended that 1–2 gallons of blood be given to the adult cow and that it be given slowly to prevent cardiac embarrassment (3). It has been reported (7) that since cardiac damage may be the result of this disease, transfusions should not be given if the animal is restless or severe restraint must be used.

The tetracycline therapy appeared to be effective in this case at 4–5 mg/lb for five days. The reactions seen during the administration of the blood may have been the result of the anticoagulant since several similar reactions have been seen in this clinic.

Control—The sources of infection are, 19 species of ticks, nine species of horse flies, deer flies, two genera of mosquitoes, and stable flies have been incriminated (9). Biological transmission by ticks has generally been considered the major source of infection, but mechanical transmission by man, biting insects such as mosquitoes and stable flies may be of considerable importance within the herd (7).

The increased incidence and/or diagnosis of anaplasmosis in non-epizootic areas is probably due to the increase in interstate shipment of cattle. Since the complete history of this cow is not known, the epidemiology could not be completed. It is likely these cattle were shipped from an epizootic area or were brought together with carrier cattle somewhere in their lives.

Few states have active control programs to regulate the disease. New York, Oklahoma and Louisiana have been the most active in trying to reduce the disease incidence and Hawaii has eliminated the disease (7, 8). These states have utilized CA and complement fixation (CF) tests, carrier animal isolation, vector control, rigid treatment methods and vaccination procedures in an attempt to control this costly disease (7, 8).

One of the most recent testing procedures developed and one that could be very useful in the field is a rapid card agglutination test (1). Vaccination procedures have been quite successful but are not without fault as indicated by comparisons of attenuated anaplasma organisms* and an adjuvant vaccine† (10). In a recent study (5) of several herds in which neonatal immunohemolytic anemia and icterus in calves was seen, it was found that all the dams were inoculated with the adjuvant vaccine.

It is quite evident that states such as Iowa, where anaplasmosis is not a severe problem, need to initiate some type of control program. Farmers (especially those buying cattle from epizootic areas or those of unknown origin) need to be made aware of the problem. Veterinarians must be aware of the fact that mechanical transmission by infected hypodermic needles, castrating and dehorning instruments and by blood transfusions is an important factor in the spreading of this disease. The tests available are convenient and economical and should be put to use now, before this cattle industry problem becomes bigger and more costly than it already is.

REFERENCES


* Diamond Laboratories
† Fort Dodge Laboratories