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Equine Rabies

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treatment to help remove some of the toxins in the mammary gland. This should be done as often as possible. Therapy as described above was used in the review case, however, it was not started soon enough to be effective. The prognosis for toxic mastitis is not good. If treatment is initiated early in the course of the disease, some success may be obtained. However, it usually leads to either recovery after prolonged therapy or to the "downer cow syndrome" terminating in death. In the specific case of Staphylococcus aureus toxemia, the toxic mastitis can lead very quickly to gangrenous mastitis which usually requires amputation of the teat or udder to save the animal's life for salvage slaughter. The review case developed areas of gangrene in the udder because of the Staphylococcus aureus infection. Despite rigorous therapy this cow progressed into a "downer cow syndrome" and died.

As treatment for toxic mastitis is always costly and often futile, prevention or early detection and treatment are essential. Early detection should be based on a complete physical examination rather than jumping to conclusions on the basis of suggestive signs. Prevention of toxic mastitis is similar to that for all other types of mastitis and should include good management and a dry-cow therapy program.

BIBLIOGRAPHY


Equine Rabies
by Ronald C. Emerson*
Ian McLeish, B.V.M. & S., M.R.C.V.S.†

Summary

Rabies in the horse can be obscured by atypical clinical signs. One such case is discussed which resulted in exposure to four people. The clinical signs of equine rabies are reviewed, and a differential diagnosis is discussed. The policy of including rabies in your differential when a CNS problem is observed is again emphasized.

Introduction

Veterinary students are confronted with the disease known as rabies (lyssa, hydrophobia) from the first day of their freshman year. The disease is studied from the pathological, clinical, and public health point of view and yet this disease is often forgotten to be entered into the differential diagnosis.

History

On November 28, 1972, a 5 year old male quarter horse was admitted to Stange Memorial Clinic with the owner's complaint that the horse had been colicy for 2 days. The owner last noticed his horse eating on November 24 and 25, and drinking little water. The owner also noticed that his horse was slightly ataxic when forced to move on November 27 and his general attitude was lethargic since November 24. He noticed that the mucous membranes of the mouth were ulcerated. The feces were described as being of normal consistency.

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The owner explained that this horse was one of six, but this one was the only one stabled since early September. All his horses were on the same ration of hay, corn, and bran with no changes of feed since November 1.

**Physical findings**

November 28, 1972. Upon admission the general appearance was noted to be that of depression. The temperature was 103.4°F. The respiratory and heart rates were normal. The mouth lesions described by the owner were examined. The anterior 10 cm. of the buccal mucous membranes were very hyperemic, with erosions evident on the anterior 3 cm. of the tongue. Small lesions (0.5 cm.) appeared just inside both nares. Slight ataxia in the hindquarters was evident when the horse was made to move. Ophthalmic examination was normal.

A tentative differential diagnosis was made:
1. Ingestion of irritant compounds such as phenols (eg. creosote).
2. Viral and bacterial encephalomyelitis.
4. Uremia.
5. Rabies.
6. Neoplasms of the C.N.S.

Several of the diagnoses under consideration could be eliminated or be given lower priority immediately. There was no history of any access to abnormal ingesta such as creosote. Viral encephalomyelitis was discounted as highly unlikely since late November is past the mosquito season. Since the other five horses were on a similar ration, moldy corn poisoning seemed remote. Evaluation of the patient's BUN would be needed to determine uremia (later the hematological reports showed a normal BUN of 12.0 mg%). Rabies should always be considered when any CNS problem is suspected.

November 29, 1972. The patient remained lethargic and more ataxic. The buccal ulcerations were hemorrhaging slightly. During the afternoon, the patient was found in lateral recumbency. Pulse rate was 60/min. with a body temperature of 101.7°F. Four liters of normal saline and one liter of 2½% dextrose were given intravenously and 10 ml. of thiamine (100 mg per ml.) intramuscularly.

November 30, 1972. The patient remained in lateral recumbency; therefore he was rolled every six hours to prevent pulmonary congestion. Upon examination the periorbital areas were severely swollen. It was felt that this trauma must have come from his dragging his head on the dirt floor while in lateral recumbency. The triceps muscles showed slight tremors with poor muscle tone. Though a swallowing reflex could not be elicited during the past two days, a reflex was produced today. Knee pressure over the neck yielded a pronounced grunting sound. The patient also passed a small amount of formed feces.

The owner was called for further history. He revealed that he had shot a skunk in the barn, four weeks ago, where the patient was then stabled. With virtual elimination of the other differential diagnoses, along with the new evidence, rabies was tentatively diagnosed. However, the clinical signs did not strongly support this diagnosis.

December 1, 1972. The temperature was 94.5°F. The respirations were of Cheyne-Stokes form at a rate of 30/min. The patient died in the afternoon and was immediately necropsied.

**Hematological Findings**

<table>
<thead>
<tr>
<th>Date</th>
<th>Hb (Gm%)</th>
<th>PCV (%)</th>
<th>R.B.C.</th>
<th>WBC (x10⁶/cu. m.m.)</th>
<th>Differential W. B. C.</th>
<th>SGOT (Reitman-Frankel units)</th>
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<tr>
<td>11/28</td>
<td>12.2</td>
<td>36%</td>
<td>Normal</td>
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<td>16.4</td>
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Pathological findings

No diagnostic gross lesions were evident. Most of the tissues had the odor of uremia when incised. The common findings of a thrombus and accompanying strongyles were present in the anterior mesenteric artery. Since the most common sites for bites from rabid animals are the muzzle and fetlock area, and no lesions were found on the legs, the horse most likely was bitten on the muzzle. The lesions around the muzzle could only be surmised as being self-inflicted from the severe irritation caused by the bite of the rabid animal.

Histopathology

The histopathology revealed perivascular cuffing of lymphocytes and focal and diffuse gliosis in the brain. Similar lesions were found in the pituitary gland and spinal cord.

The fluorescent-antibody test was performed on the brain tissue. It was found to be positive. Negri bodies were absent in brain section; this is a consistent finding in equines. As final confirmation, mice were inoculated and died within eleven days.

A cerebrospinal fluid tap was performed just prior to necropsy. Pandy test 3+; glucose 60 mg%; 57 cells/cu. m.m.; all lymphocytes. A urinalysis was also performed. Urinalysis: color—light yellow; specific gravity—1.036; pH—6.0; protein—30 mg%; acetone—negative; sugar—4+; blood—large; bilirubin—conjugated—negative; urobilinogen—1.0 Ehrlich units/100 ml. urine; and sediment—amorphous material. The abnormally high sugar in the urine was explained by the fact the urine sample was taken during necropsy, after the adrenals had raised the blood sugar above the renal threshold. The large amount of blood could be explained by the technique of getting the urine sample at necropsy by bladder puncture. Culture of CSF and urine were both negative.

Discussion

Two aspects of rabies in the horse must be emphasized. One, though only about 40 cases of rabies are reported in the horse each year in the United States, rabies should be suspected anytime a CNS problem is evident. Rabies was not highly suspected in this case, consequently human exposure occurred when examining the mouth. Two, the clinical signs as shown by this case are not necessarily typical as compared to most reported cases.

The clinical signs commonly reported include early signs of hyperesthesia, hyperexcitability and viciousness, inflammatory reaction at the site of bite inflicted by the rabid animal, later tremors and muscular spasm leading to hindlimb paralysis and finally convulsions. The pulse rate and respiratory rate are increased from the onset of clinical signs until death.

Note

Three of the four people exposed had already received rabies vaccine periodically during veterinary school and had antibody titers. The question immediately arose whether or not they needed to undertake the 14 day rabies vaccination series. Physicians at the university hospital felt that in any incidence of positive exposure to rabies the 14 day rabies series was indicated regardless of previous vaccination history or antibody titer. Their explanation for rabies vaccination prior to exposure is for protection of the individual from unknown exposure.

REFERENCES