Polyradiculoneuritis of the Coon Hound (Coon Hound Paralysis)

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Case Report

A tan coon hound was referred to Stange Memorial Clinic at Iowa State University on February 14 in a tetraplegic state. The hound had been hunted for 12 days prior to a fight with a raccoon on February 7. In his courageous attempt to kill the raccoon with a steel pipe, the hunter accidently hit the hound on the head. At this time the dog seemed to be uninjured, but three days later was found to be unable to rise. The animal was then treated with systemic antibiotics, corticosteroids, and aspirin by the referring veterinarian. The paralysis continued to progress anteriorly, and the hound was referred to Stange Clinic four days after the onset of clinical signs. At the time of our examination, the hound was alert and tetraplegic, being able to move only the head and neck. The muscles of the limbs were very soft and atonic. The pupillary reflex was present, but slow. Radiographs of the cranium revealed no abnormalities.

The differential diagnosis included coon hound paralysis, rabies, cerebral edema, and subdural or extradural hematoma. On the basis of the history and flaccid tetraplegia, lower motor neuron damage resulting from coon hound paralysis was considered the most probable diagnosis.

Supportive and symptomatic treatment was initiated. A very palatable high protein diet* was fed by hand. The dog usually ate well if the food was placed in its mouth. A solution† of procaine penicillin G in dihydrostreptomycin sulfate with prednisolone was given at the maximum dose twice a day. Water intake was usually normal, but dehydration developed by the fourth day of treatment. This was corrected with 250 cc. of a protein hydrolysate solution‡ given intravenously. Urination and defecation remained normal. A wire platform was placed under the dog to prevent urine contact.

Profound atrophy of the appendicular musculature was evident by one week

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* p/d Prescription Diet, Riviana Foods Inc., Hills Division, Professional Products Dept., Topeka, Kansas.

† Bio-Delta, The Upjohn Company, Kalamazoo, Michigan.

‡ Ambex, Elanco Products Company, Division of Eli Lilly and Company, Indianapolis, Indiana.

after the onset. The forelimbs became very edematous from inactivity. After two weeks, decubital ulcers began to develop over the lateral prominences of the skeleton (acromion, greater trochanter, and lateral epicondyle of the humerus). At the end of four weeks, muscular atrophy and decubital ulceration were very severe, but there was still no relief from paralysis. The hound was euthanized at this stage at the request of the owner.

Unfortunately, only gross necropsy findings are available. The cranium was normal and intact. The meninges and brain were normal grossly. The fluorescent antibody check for rabies was negative.

Discussion

Little work has been done on polyradiculoneuritis of the coon hound, and the etiology is still a matter of personal speculation. Both spinal trauma and intoxication with *Clostridium botulinum* toxin have been proposed, but solid evidence for either is lacking. A similar paralytic disease of man, the Landry-Guillain-Barre' syndrome, is thought to be of allergic origin. Regardless of the direct etiologic agent, contact with a raccoon seems essential.

The paralytic hound is usually admitted to the clinic with a history of a fight with a raccoon 7–14 days previously. Paraparesis progresses rapidly to flaccid tetraplegia. The animal usually remains alert through the course of the disease. In the acute stage, there may be severe respiratory depression probably due to partial paralysis of the respiratory muscles. Affected dogs often have a weak and peculiar voice. Recovery from paralysis in 3 to 4 weeks is most common; however, some dogs require several months for recovery.

The characteristic flaccid paralysis suggests damage on the level of the lower motor neuron. The severe and rapid muscle atrophy is similar to that seen following peripheral neurectomy. Histological examination of spinal cords from affected dogs reveals demyelination, axis cylinder degeneration and central chromatolysis of the ventral horn cells. On the basis of histopathologic findings, the name polyradiculoneuritis seems very appropriate to describe the disease.

Treatment of polyradiculoneuritis of the coon hound is supportive and symptomatic. The use of systemic antibiotics is indicated as a prophylactic measure for the debilitated animal. Some report that early use of corticosteroids will reduce the progression of the paralysis. Actually, any therapeutic effort is difficult to evaluate due to the natural variability of the recovery time in this disease. Some dogs may succumb in the acute stage due to respiratory embarrassment unless a respirator is used. The use of an air mattress, regular turning of the dog, and massage will minimize decubital ulceration.

After a variable period of time, motor function will slowly return in reverse to the order in which it was lost. The muscles should be massaged and exercised for short periods several times a day. Most dogs overcome the muscular degeneration to enjoy a near normal recovery. In long standing cases marred by severe decubital ulceration and muscular wasting, euthanasia should be considered in view of the suffering and improbability of a complete recovery.

Conclusion

Coon hound paralysis is not real common in Iowa, but it should be considered in many differential diagnoses involving lower motor neurological disease. Because the paralytic phase of rabies mimics polyradiculoneuritis in many ways, one should always consider the possibility of rabies when examining an animal that appears to have coon hound paralysis. Treatment of this disease is laborious, time consuming and not very gratifying. On this account, the veterinarian may do well to send the affected animal home after the acute stage of the disease.

REFERENCES

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3. Kirk, R. W.: Current Veterinary Therapy III Small Animal Practice, W. B. Saunders Company, Philadelphia, Pennsylvania. 1968.