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Equine Leukoencephalomalacia

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Summary
The clinical course and post mortem findings of a case of Equine Leukoencephalomalacia are discussed. Some of the more important aspects of Leukoencephalomalacia in the equine are also reviewed.

Introduction
This report describes a case of leukoencephalomalacia in a horse which was admitted to the Iowa State University Teaching Hospital. The case is used as a vehicle to review some of the more important aspects of LEM.

Clinical History
On July 15, 1978 a three year old Morgan mare was presented to the Iowa State University Teaching Hospital for treatment of a laceration on the right rear leg.

On July 22nd the horse was seen to be depressed and when forced to move would stumble forward as if it were unable to catch its balance. On the 23rd the horse still would not move and was placed on antibiotic therapy. On the 24th the horse was down in its stall but on the 25th it was up again. Although it was still depressed, it was somewhat improved and improvement continued through July 31st.

On August 1st the horse again became weak, depressed and ataxic. Antibiotic therapy (procaine penicillin) was reinstituted but the horse continued to worsen through August 5th when a partial left facial paralysis was noted. The horse’s condition then began to improve until August 16th when its condition again worsened. On August 17th the horse died.

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Treatment
The horse was treated with 22 cc of Pen/Strep intramuscularly on July 23rd and 22 cc of procaine penicillin daily from July 24th through July 27th. On August 1st, 10 cc Vit. E Selenium and 20 cc procaine penicillin were given. The penicillin therapy was continued through August 6th and butazolidin was also given on August 3-6.

Clinical Pathological Findings
On August 1, 1978 hematological examination of a peripheral blood sample revealed a WBC of 12,200/mm³ with 5% banded neutrophils, 80% segmented neutrophils, 12% lymphocytes, 3% monocytes, and the presence of toxic neutrophils. The hematocrit was 39%, plasma protein 7.6 g % and fibrinogen 100. SGOT was 613.7 IU/L compared to normal of 50 (range 0-200) for the ISU clinical pathology laboratory.

Pathological Findings
At necrospy an extensive area of hemorrhage and malacia was present in the thalamus. The lesions were bilateral and extended slightly into the white matter of the cerebral cortex.

Microscopically there was extensive hemorrhage and thrombosis in the thalamus. The white matter of some of the cerebral folia displayed acute malacic change. Sections of the spinal cord showed no gross or microscopic lesions.

Discussion
Leukoencephalomalacia (LEM) is a consistently fatal disease of horses and other Equidae characterized by destruction of portions of the white matter of the brain. Although the disease has been reported in the United States since 1850, much remains to be discovered about it. Only a very limited amount of controlled research has been published and most of the available literature...
consists of descriptions of the lesions and clinical course of the disease. These two aspects of the diseases have been well characterized and numerous authors spanning four decades are in remarkable agreement on these matters.

The clinical signs most commonly attributed to LEM are weight loss, ataxia and depression. A wide range of other neurological signs have been reported in cases of LEM with varying frequency. These include facial paralysis, drowsiness, and twitching of the muscles of the shoulders and thighs among others.

Gross pathological lesions have been described extensively and consist mainly of an intact cortex overlying a cerebral lesion which is soft and edematous. Some of the lesions have been reported to have extensive hemorrhage in and around them. Although the lesions have been reported most frequently in the cerebral cortex they have been reported in the thalamus, cerebellum and brainstem. The lesions have been reported as unilateral as well as bilateral.

Changes have also been occasionally mentioned in nearly all of the other organ systems. These include, for example, swollen and congested liver and kidneys as well as erosions of the GI mucosa. There are several other reported cases in which no gross or microscopic lesions were found.

The microscopic lesion has been reported nearly unanimously as liquefaction necrosis. While the published descriptions of the lesions and clinical course of the disease are legion, very little is known concerning the pathogenesis and etiology. The preponderance of evidence points to Fusarium moniliforme as the etiologic agent. The toxic principle has not been identified and the establishment of F. moniliforme as the etiologic agent is less than absolute. Although it has been established that F. moniliforme was present on corn that caused the disease, it has never been established that corn contaminated only with F. moniliforme can cause the disease. In one trial three types of fungi were isolated from corn which produced the disease and F. moniliforme on corn was fed to a mule and did produce the disease. When further trials were attempted with this same isolate the disease was not produced. It was speculated at that time that, "the mold had lost its toxigenicity."

In the few trials where the disease was produced by feeding moldy corn, periods varying from slightly less than two weeks to as long as several weeks elapsed from the commencement of ingestion until clinical signs appeared.

Most cases of LEM in the United States have been reported either in the fall when a dry growing season was followed by a wet period or in November through May of the following year. This fact seems more likely to be associated with management practices concerning when the horses have access to the contaminated corn rather than to the disappearance of the etiologic agent once it has appeared.

The facts that are known about LEM are compatible with a mycotoxin as the etiologic agent. It is well known that mycotoxins of Fusarium spp. are elaborated only when a very specific set of conditions exist. As has been recently illustrated with aflatoxin, the presence of the fungus alone means nothing. It is possible that a toxin or a toxin metabolite of F. moniliforme is indeed the etiologic agent but many other possibilities also exist. Soon to be published work by Dr. M. Abou-Gabal has produced a definite link to encephalomalacia in rabbits.

While the etiologic agent is not known, "foci of liquefaction necrosis in the white matter of the brain either in one or both hemispheres is considered to be pathognomonic for LEM." Should the etiologic agent be found, this statement may well have to be modified, but at present it poses no problem. A diagnostic problem does exist, however, in those cases which do not clearly have this pathognomonic lesion. As has been mentioned previously, fatal cases have been reported with other lesions or with no lesions in the brain at all. The possibility that some of these reported cases were not LEM at all clouds the issue but is not grounds to dismiss them. The case presented here is illustration of the uncertainty that exists in some cases.

This horse began showing signs seven days after hospitalization in July. This is not the typical time for LEM to appear. The location of the lesion was not typical although previously reported. Areas of liquefaction necrosis were present as were areas of...
hemorrhage and thrombosis. These are all compatible with LEM as noted previously. The etiologic agent could have caused the hemorrhage and thrombosis as well as the liquefaction necrosis but a cerebrovascular accident could also have been the cause.

The pathologist's diagnosis in this case was that LEM was the most probable cause of death but that a CVA could not be ruled out.

References

8. Personal communication.

SEATTLE, WA.—Eight veterinarians received awards during the 116th annual meeting of the American Veterinary Medical Association (AVMA) in Seattle, Washington. The awards were presented at the Inaugural and Awards Luncheon on Tuesday, July 24.

*AVMA Award*—Dr. Frederick D. Wertman, Jr., Des Moines, Iowa. The award is given annually to a veterinarian in recognition of distinguished contributions to the advancement of veterinary medical organizations. Dr. Wertman graduated from Iowa State University in 1940 and conducted a general practice in Carlisle, Iowa, until 1960, when he became executive director of the Iowa Veterinary Medical Association. He also currently serves in the same capacity with the American Association of Swine Practitioners and as president of the National Board of Veterinary Medical Examiners.

AVMA News Release